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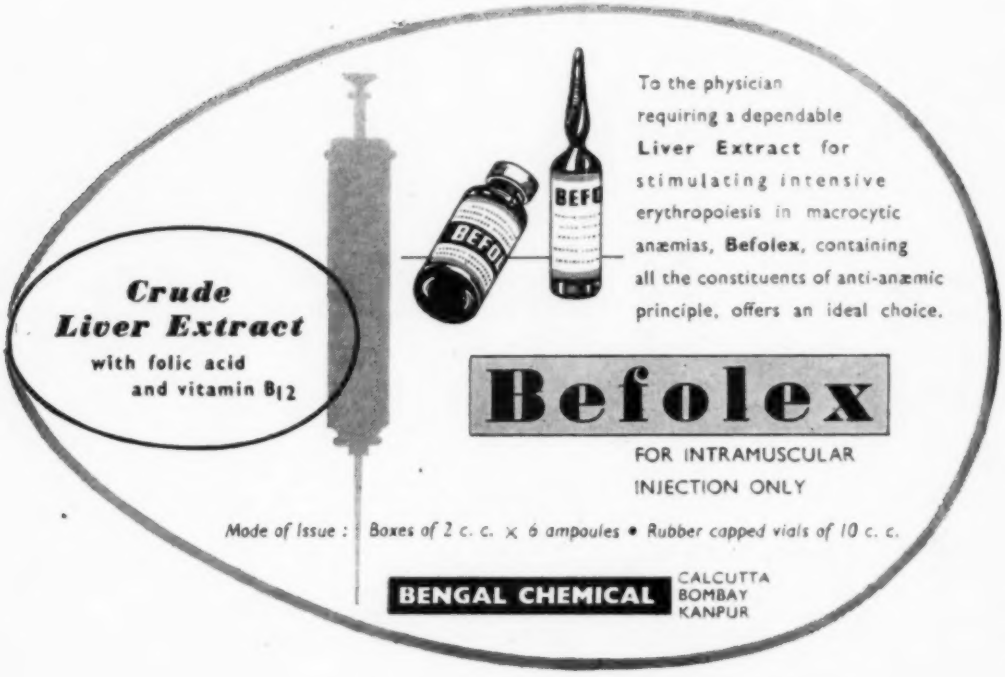
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JOURNAL OF THE INDIAN MEDICAL ASSOCIATION

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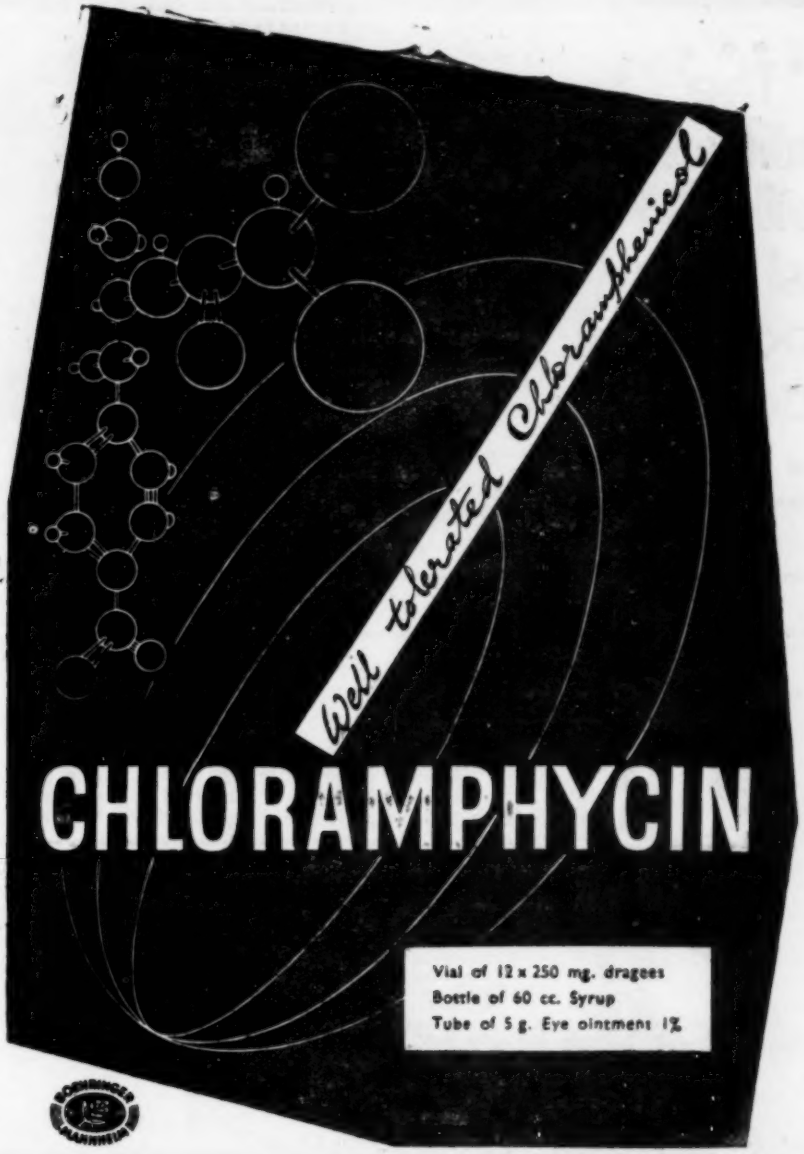

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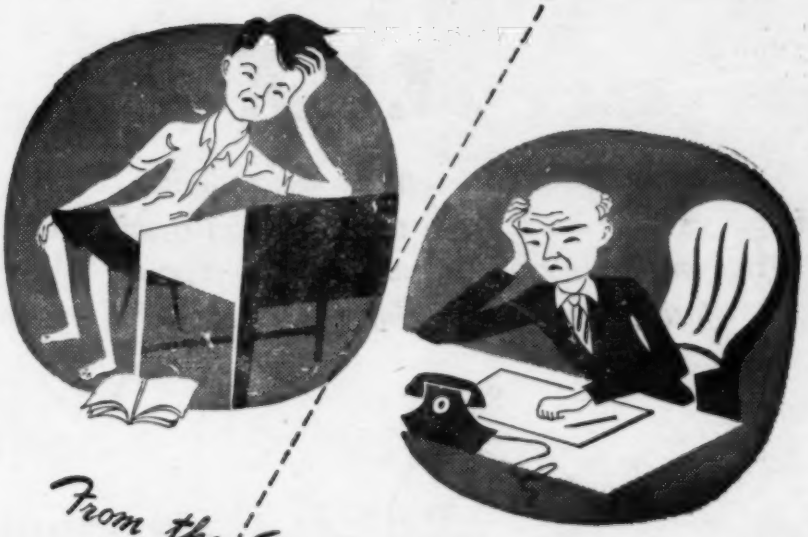

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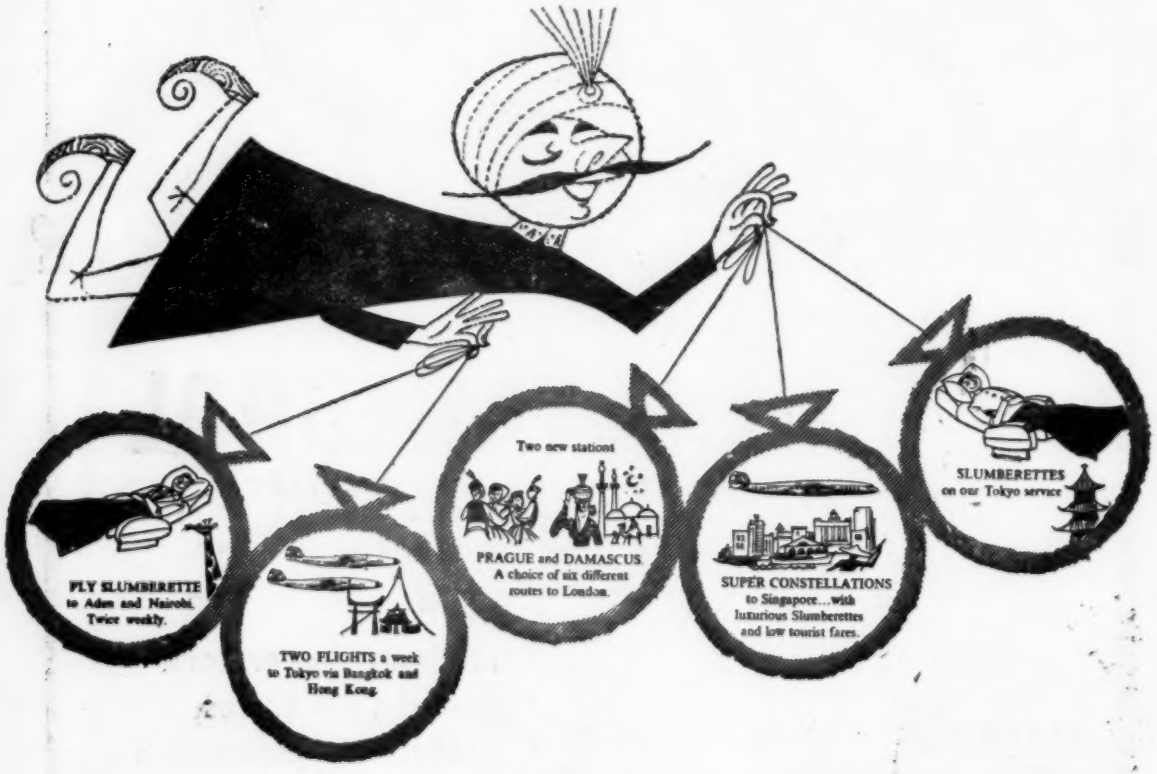
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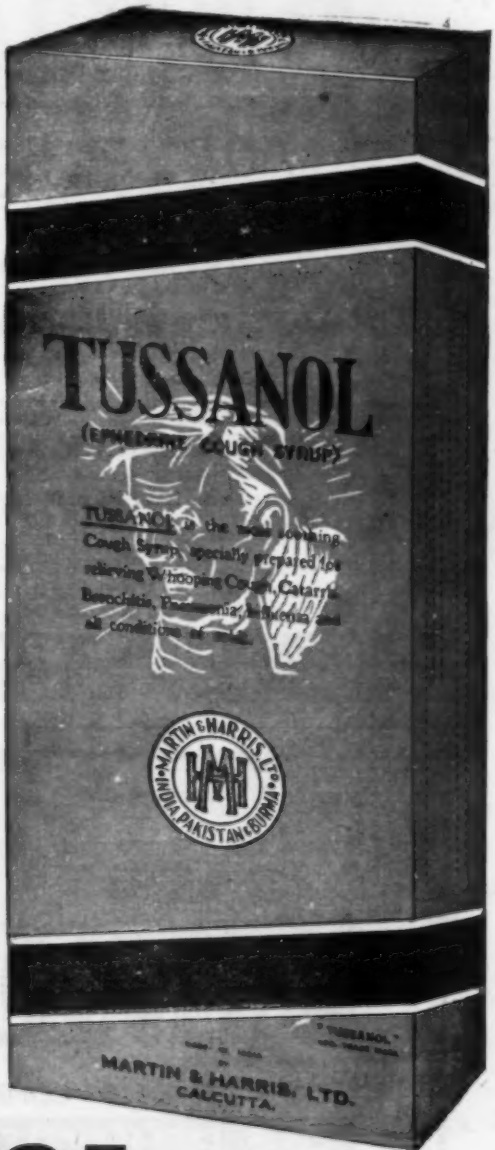

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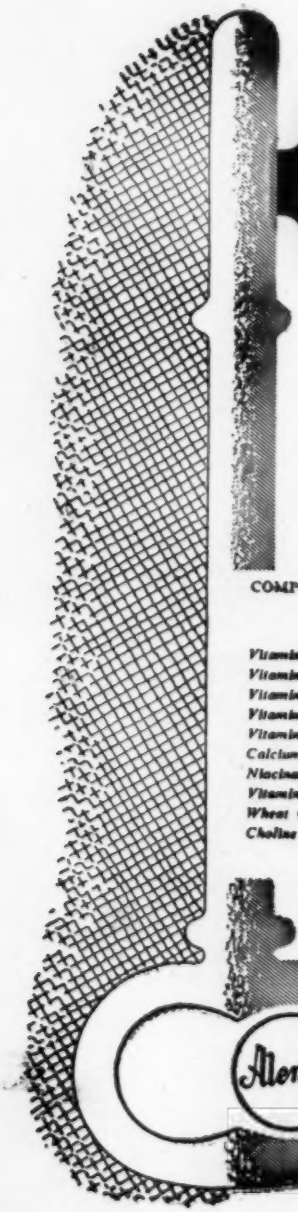

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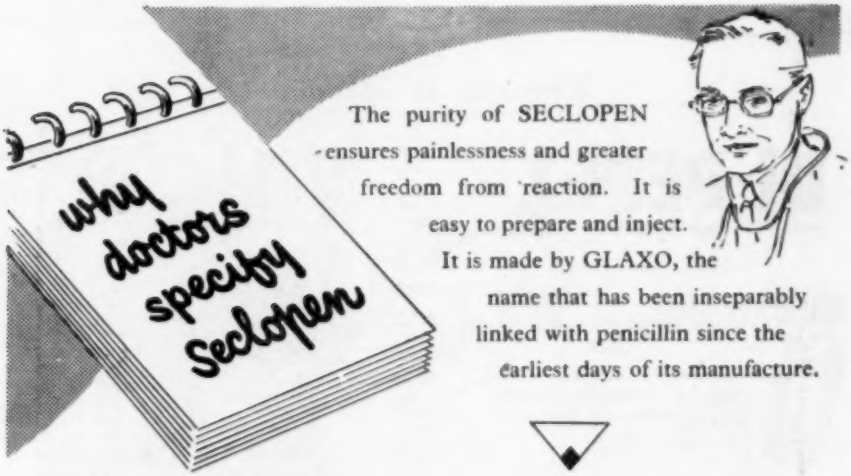

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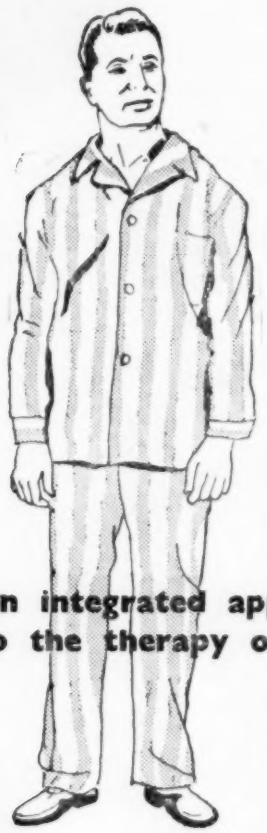

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ORIGINAL ARTICLES

INFESTATION WITH OVA MORPHOLOGICALLY RESEMBLING *SCHISTOSOMA HAEMATOBIUM*

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The four cases here reported have been encountered between the dates 29-3-55 and 7-10-55 during routine examination of the stools. Though one of the cases has been referred from another laboratory, three of them are among approximately 500 routine stool examinations done between the dates mentioned. It would seem from these facts that though cases of schistosomiasis have been reported occasionally from different parts of India, and one definite focus has been discovered by Gadgil and Shah (1952) in a village in Bombay State, this disease may actually be much more prevalent than has been formerly supposed.

Case Reports

Case 1—Mrs. M., aged 55 years, belonging to the middle income group, was born in Bhon (West Punjab) and migrated to Delhi after partition. She complained of flatulence, palpitation and sinking feeling on 11-2-55. She appeared to be well-nourished and not obviously anaemic. Clinical examination elicited no positive finding. Routine stool examination was done on 8-3-55, when cysts of *E. histolytica* and *E. coli* were found. After one course of treatment for the intestinal amoebiasis, she came for a check up of the stool on 29-3-55 when she said she felt much better. No intestinal protozoa or cysts were found. Mucus was present mixed with the stool and pus cells in fairly large numbers, and a few R.B.C. were found. Ova with terminal spines morphologically resembling those of *Schistosoma haematobium* were also detected (Fig. 1, *vide* Plate). Their number was about one to three per cover-slip preparation without concentration of the stool. The ova were fertile and mostly mature specimens. The urine was examined repeatedly but no ova were found. Ova were found in the stools on two other occasions.

Case 2—Mr. C., belonging to the higher income group, has lived mostly in Delhi, Dalhousie and Lahore and has visited the Kulu Valley, Dharamsala and Kashmir for fairly long periods. Very few facts are known about this case, as since the discovery of terminal-spined ova in his stool in July 1955 he has not been available for questioning and further laboratory examinations. The ova were initially detected in the stool specimen in a different laboratory. In subsequent examination of the same specimen, one to four mature and fertile ova were found per cover-slip preparation (Fig. 2, *vide* Plate).

Case 3—Mrs. Ch., aged 29 years, was born in Amritsar and spent fairly long periods in Simla, Dalhousie, Mussoorie and Kashmir and is now settled in New Delhi. She belongs to the high income group. On 8-8-55 she complained of nervousness, irritability, aching of arms and legs, insomnia and repeated attacks of alternating diarrhoea and constipation from which she had suffered off and on for six years. She complained of painful and difficult micturition recurring at fairly frequent intervals. She was somewhat overweight and slightly anaemic. The only clinical finding was a large cystocele. Routine stool and urine examinations were done on 10-8-55. Ova with terminal spines, morphologically resembling those of *Schistosoma haematobium*, were found in the stools (Fig. 3, *vide* Plate). The ova in this case did not appear to be mature. No ova were found in daily examination of the urine for a period of two weeks. No further ova were found in the stools either, during that period. Cysts of *Giardia intestinalis* were present in large numbers.

Case 4—V. K., male, aged 17 years, was born in Jammu. He is the son of a government servant of medium grade who has been posted in many places in Punjab (I) and also in Kashmir. He had been examined by a medical board on 11-2-55 and declared temporarily unfit on account of anaemia. His erythrocyte count was approximately 3 million per c.mm. and his haemoglobin

was 13 g. per cent. Clinical examination was negative except for anaemia. Routine stool and urine examinations were done on 7-10-55. One terminal-spined ovum was found in the second slide during the stool examination. On concentration of the stool one to four ova were found per slide. Some of the ova appeared to be fairly mature and fertile, others were immature (Fig. 4, *vide* Plate). No ova were found in the urine. No radiological evidence of schistosomiasis was found in the urinary bladder.

Discussion

From the above case reports several facts emerge :

1. These cases have all been discovered accidentally on routine stool examination over a period of about six months.
2. None of them gave histories in any way relevant to urinary schistosomiasis or exhibited any of the signs or symptoms thereof.
3. In a limited and varying number of examination no ova were found in the urine.
4. None of the cases are from the lower income groups, very few examinations in the total of 500 being from these groups.
5. None of the cases have lived South or East of Delhi (Table 1) except one who has visited Mussoorie (in a meridian slightly East of Delhi), and one who has lived in Gurgaon (slightly South of Delhi).
6. Research in this subject seems to be indicated. It appears to be possible that apart from the institution of organised research, if medical practitioners were alerted to the possibility of the occurrence of schistosomiasis in India, more facts would come to light.

Table 1

Case No.	Places of residence	State
1.	Bhon-Tehsil Chakwal, District Jhelum	Punjab (P)
	Gujjarkhan	Punjab (P)
	Lodi Road, New Delhi ...	Delhi
*	Tilak Nagar	Delhi
2.	Lahore	Punjab (P)
	Dalhousie	Punjab (I)
	Kullu	Punjab (I)
	Dharamsala	Punjab (I)
	Kashmir	Kashmir
	Delhi	Delhi
3.	Amritsar	Punjab (I)
	Simla	Punjab (I)
	Dalhousie	Punjab (I)
	Mussoorie	Uttar Pradesh
	New Delhi	Delhi
	Kasauli	PEPSU
4.	Jammu	Kashmir
	Srinagar	Kashmir
	Hoshiarpur	Punjab (I)
	Kullu	Punjab (I)
	Garhshankar	Punjab (I)
	Panipat	Punjab (I)
	Karnal	Punjab (I)
	Jullundur	Punjab (I)
	Moga	Punjab (I)
	Dasuya	Punjab (I)
	Gurgaon	Punjab (I)

Summary

Four cases are reported where ova with terminal spine, morphologically resembling those of *Schistosoma haematobium* were encountered during routine stool examination of 500 cases. None had any specific symptom for this infestation and there were no ova in the urine.

Acknowledgment

My thanks are due to Dr. J. S. McKenzie Pollock, W.H.O., New Delhi, for encouragement and advice, to Dr. K. R. Joneja, Delhi, for allowing me to examine the stool specimen of Case 2, and include it in this series, and to Dr. D. Sen, PH.D., National Physical Laboratory, New Delhi, for the photographs.

Reference

Gadgil, R. K. and Shah S. N.—*Indian J. M. Sc.*, **6**: 760, 1952.

HISTAMINE SHOCK IN ADRENALECTOMISED RATS

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In a large series of experiments it was found that histamine is ineffective in the production of gastric ulcer in rats (Basu Mallik, 1955). During this work a report by Selye (1936) came to the notice of the author. In the communication Selye reported that intraperitoneal injection of 20 mg. of histamine into adrenalectomised rats can cause acute gastric ulcer. It was therefore decided to investigate the matter.

Method and Material

Wistar strain of albino rats weighing between 120 and 180 g. were used in this experiment. Dorsal route of approach of the suprarenal glands was selected. Under ether anaesthesia and with

proper aseptic precaution a midline incision was made dorsally, extending from the lower thoracic vertebra to the sacrum. After the separation of the skin the lateral margin of the spinal muscle mass (sacrospinalis) was identified and incised. The upper pole of the kidney was thus exposed and the adrenal identified. The pedicle of the gland was then found out and a mosquito forceps was clamped to it. The pedicle distal to the clamp was cut and the gland was lifted up and removed. The clamp was kept for two minutes to check bleeding and then removed. During the whole procedure care was taken not to hold the gland by the forceps as such manoeuvre would cause squashing of the gland substance. The same procedure was repeated on the other side. The wound was closed by a double layer of sutures. The animals were given food *ad lib.* and were allowed to drink water containing 1 per cent sodium chloride. They made uneventful recovery though they appeared to be rather weak after the operation. One week after the operation histamine (20 mg. of the base) was injected intraperitoneally.

Result

Soon after the injection the animals became very weak, and looked ill, sitting quietly with arched back and hairs standing out. They had respiratory difficulty. Within 10 minutes they lost consciousness and most of them died within 15 to 60 minutes. Those that did not die within one hour were killed and autopsy performed immediately afterwards.

The abdominal cavity of these animals contained some serosanguinous fluid. All the abdominal viscera were intensely congested. Stomach, when opened up, showed marked congestion of the mucous membrane, specially of the glandular part. There was no obvious ulcer. Microscopically, capillaries and veins were found to be engorged with blood. There was no ulceration or any sign of inflammation nor any collection of inflammatory cells. That the congestion was due to passive hyperaemia was evident from the fact that although the capillaries and the veins were markedly congested, the arteries and arterioles remained bloodless and collapsed (Fig. 1, *vide* Plate). The appearance is very similar to that of portal venous obstruction. No marked secretory activity of the gastric glands could be demonstrated histologically by special stains, viz., for pepsinogen granules and for mucin. Similar congestion was noticeable in the intestine and the liver.

Discussion

Histamine shock in adrenalectomised rats failed to produce any gastric ulcer though it was responsible for marked venous stasis in the stomach. Therefore Selye's observation could not be confirmed. In his communication Selye did not give any description of the ulcer nor did he mention the histological appearance of these ulcers. It is possible that he made only macroscopic examination, which is often very deceptive as we have found to our dismay. The common mechanism of the genesis of gastric ulcer is either by the peptic digestion of the devitalised mucosa or by ischaemic necrosis. Removal of adrenal glands in rat reduces the secretion of the gastric juice (Tuerkischer and Wertheimer, 1944). Though histamine is a powerful stimulant of the oxyntic cells in other species of animals, it stimulates only mild secretion of acid in rats (Basu Mallik, *loc. cit.*). So the possibility of acid digestion of the mucosa is remote after the injection of histamine in this species. The intense capillary engorgement that was observed in these animals rules out the possibility of localised areas of ischaemia. It is therefore not difficult to envisage why ulceration does not occur in adrenalectomised rats after the injection of histamine. Removal of adrenal glands may result in gastric ulceration in some species of animals, e.g., dog (Mann, 1916). The stomach of several rats was examined one week after adrenalectomy and in none of the specimens was there any evidence of gastric ulcer. As to the causation of the severe venous stasis, it is apparently due to the spasm of the hepatic vein which can explain the venous congestion of liver and the portal system. Injection of toxic dose of histamine in dogs has similar effect on the hepatic vein.

Although the rat is highly resistant to the toxic action of histamine (Voegtlin and Dyer, 1924), removal of both the adrenals makes this animal extremely susceptible to the noxious effect of histamine (Crivellari, 1927; Marmorston-Gottesman, 1928). This is because of the fact that in the normal rat, secretions of the adrenal gland help in the detoxification of histamine (Best, 1929). Bilateral adrenalectomy may thus make many species of animals susceptible to shock where histamine or a similar substance is liberated.

In recent years adrenalectomy is being performed for various maladies like breast cancer, hypertension etc. Post-operative shock in some of these patients may cause severe venous stasis in the portal bed and may even lead to severe gastric haemorrhage. I had occasion to examine the stomach of two such patients who had severe

haematemesis after bilateral adrenalectomy. The histological appearance of the organ in these patients was similar to that found in the stomach of adrenalectomised rats after the injection of histamine.

SUMMARY

Injection of histamine into adrenalectomised rats may lead to fatal shock with venous stasis in the stomach but it does not result in gastric ulceration.

Significance of this finding has been discussed.

ACKNOWLEDGMENT

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REFERENCES

BASU MALLIK, K. C.—*J. Path. & Bact.*, **70** : 549, 1955.

BEST, C. H.—*J. Physiol.*, **67** : 257, 1929.

CRIVELLARI, C. A.—*Am. J. Physiol.*, **81** : 414, 1927.

MANN, F. C.—*J. Exper. Med.*, **24** : 329, 1916.

MARMORSTON-GOTTESMAN, J. AND GOTTESMAN, J.—*Ibid*, **47** : 503, 1928.

SELYE, H.—*Canad. M. A. J.*, **34** : 706, 1936.

TUERKISCHER, E. AND WERTHEIMER, E.—*J. Endocrinol.*, **4** : 143, 1944.

VOEGTLIN, C. AND DYER, H. A.—*J. Pharmacol.*, **24** : 101, 1924.

DECORTICATION IN CLOTTED HAEMOTHORAX

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There comes a stage in the management of haemothorax when the extravasated blood in the pleural cavity can no longer be satisfactorily aspirated and the underlying lung is seen to be still unexpanded on account of a layer of clotted and organizing blood. Left as such, this organizing layer forms a dense fibrous capsule beneath which the lung is captive, and, restricted in respiratory excursion, finally leads to a contracted haemothorax with considerable reduction in vital capacity.

PATHOLOGY

Blood in the pleural cavity, whether from intercostal vessels or parenchymal in origin, is now known to clot at once. Thoracic surgeons are familiar with the jelly-like clots in the pleural cavity following a sudden collection of blood during a thoracotomy. Cardiac and respiratory movements soon defibrinate these clots to liberate strands of fibrin which sink to the bottom of the cavity or adhere to the surface to form multiple pockets. Almost within twelve hours following injury this defibrinated and partially haemolysed serum excites a marked irritative reaction from the pleura resulting in further fluid exudation within the space.

Secondary clotting occurs at this phase, aided undoubtedly by thromboplastins from lacerated lung and, sometimes, the presence of mildly infective organisms. Serum, blood cells, fibrin, and tissue elements combine to form the contents of the haemothorax. Fibrin is deposited all over : on the visceral surface of the lung, in the floor of the cavity and peripherally on the inside of the parietal pleura. Within this fibrinous cortex, liquefaction occurs. Adherent bands, entrapped air, form multiple pockets ; and if the infective element is strong, empyema results. Aspiration becomes difficult and enzymic therapy with fibrin solvents may not succeed.

Not all haemothoraces clot in this fashion. There is a difference between a simple traumatic haemothorax and the compound one with complex damage to intrathoracic structures necessitating urgent surgical intervention. Compound cases seldom give any further trouble, once actively tackled, and the majority of the simple haemothoraces (almost 90 per cent) respond to aspiration therapy alone.

Aspirations must start as soon as possible after the injury, must be as complete as possible, aseptically performed, and concentration devoted throughout on an intensive programme of breathing exercises to get full re-expansion of the subjacent lung. The fear of aspirations provoking further haemorrhage is unfounded ; it must not deter early aspiration. Air replacement of aspirated blood is illogical ; active expansion is prevented, and not uncommonly a pyopneumothorax results. It is only in the small percentage where aspirations fail to empty the cavity and secure a good lung expansion that clotted haemothorax is deemed to have taken place.

Often, when the full pathological evolution of a condition is not fully understood, description compromises with a term that is comprehensive yet non-committal, and if the expression is at all

attractive, easy to use or has the support of tradition, it gains wide acceptance. Clotted haemothorax, as the term denotes, does occur in a few cases. Here, the initial blood lost does not difibrinate and liquefy after primary clotting but proceeds directly to fibrous organization of the clot. In quite the greater majority of cases the condition should be called clotted haemofibrinothorax, since it represents the secondary fibrinous clotting of the exudate after liquefaction of the initial clot. Clotted haemothorax, however, remains the shorter and more favourite term of the two.

Treatment

Clotted haemothorax is not a very common condition. While the majority of simple haemothoraces are cured by aspiration therapy alone, and about 30 per cent are also complicated by infection, only about 10 per cent become clotted haemofibrinothoraces, as defined above.

The evolution of a treatment is often dependent on an understanding of the surgical pathology of that condition. Though an early clotted haemothorax can be treated simply, by thoracotomy and evacuation of the fluid and fibrinous matter, this fails to remove the enveloping fibrous cloak over the lung and under the chest wall. Pulmonary decortication is being used since a long time as a surgical procedure for the treatment of chronic empyema ; but an understanding in the development of the clotted haemothorax of the relationship of this fibrinous cortex to the pleura, and recognition of the time sequence of these pathological events, were rather delayed. Samson and Burford (1943) began reporting their results of total pulmonary decortication for clotted haemothorax. With modifications in the technique of operation and improvements in post-operative management, pulmonary decortication now gives a high rate of adequate re-expansion of the lung. The best time would be about six weeks after the injury, but haemothoraces have often not been decorticated till the third to sixth month, in which case, of course, the procedure is rendered more difficult.

Usually the sixth rib is resected with the patient in either face down or lateral position. General anaesthesia is used with positive pressure inflation through an intratracheal tube. Good wide access is desirable. The parietal layer of the cortex is incised and contents of the fibrinothorax evacuated. The pulmonary cortical layer is now carefully incised till the underlying lung, seen shining through as bluish tissue, is exposed. With small gauze pledgets caught at the tip of strong forceps like Moynihan's cholecystectomy forceps, this pulmonary cortical layer is peeled off, carefully maintaining the plane of cleavage throughout to minimise laceration of the lung. Stripping is continued till apex and base are freed, the lung visibly expanding with positive pressure as the fibrous coating is removed. The mediastinum is not usually coated heavily, but the diaphragm and parietal coating must also be removed if full expansion is to be obtained. This last manoeuvre is essential : the parietal coating being much thicker than the pulmonary, the chest wall cannot expand until this parietal layer is also removed.

Post-operative—Multiple small lacerations on the lung surface will leak air, and the dissection of the parietal layer through the extrapleural plane is bound to cause some oozing, sometimes amounting even to a troublesome haemorrhage. Two drainage tubes, one apical and one basal, connected to a water-seal bottle, will usually look after these. Attaching a suction apparatus 48 hours later in the post-operative period will help satisfactory expansion, but no mechanical effort succeeds so much as the effect of active chest expansion by inspiratory effort and breathing exercises carried out from the very next morning. As soon as the tubes are out, which is generally by the fifth day, if all leaks have stopped, the patient is got out of bed and walked about. Later, more strenous gymnastic class work should be started, the result in a few weeks being a startling difference between the dull motionless and opaque chest, and an actively expanding and radiologically clear lung.

Case Report

N.M., aged 43, had a fall from a bullock cart and was run over by one wheel on 1-4-55. He was unconscious for a short time. He was admitted to N. R. S. Hospital on 4-4-55. On examination he had a closed fracture of the 6th, 7th and 8th ribs of the left side with chest abrasions. A thin generalised opacity demonstrable in x'ray was present at this time (Fig. 1, *vide* Plate). The fractured side was strapped and active breathing encouraged. Later, aspiration was attempted but failed to draw much fluid. The temperature persisted around 101°F. On 17-4-1955 decortication was performed, with face down by 6th rib resection. Extrapleural separation of parietal cortex from apex to base was followed by entry into haemothorax and evacuation of contents. Incision of pulmonary cortex and stripping of lung surface were made avoiding areas of strong adhesion where the original lung laceration had occurred. The peel came off satisfactorily and good lung expansion was obtained. Parietal cortex was also removed. Haemorrhage from the extrapleural plane was not very troublesome. The wound was closed with drainage—apical and basal. The latter was removed at 48 hours, and suction on apical tube maintained for further two

days. Active breathing exercises and long walks were encouraged. Temperature came down to normal the morning after operation and stayed down. Radiogram 3 weeks after operation showed well expanded lungs (Fig. 2, *vide* Plate). The patient was discharged from hospital on 31-4-55.

SUMMARY

Clotted haemothorax is more a problem of war and riot injuries than civilian accidents. Almost 10 per cent of haemothoraces proceed to clotting, about 30 per cent become infected haemothoraces.

Decortication is indicated when either aspirations fail to clear the collection, or multiple pocketing and clotting render aspiration a technical failure.

Correctly speaking the term should be haemofibrinothorax.

The technique and points in postoperative management are briefly described with a case report.

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REFERENCE

SAMSON, P. C. AND BURFORD, T. N.—*J. Thoracic Surg.*, **16**: 127, 1947.

CAVERNOUS SINUS SYNDROME IN PITUITARY TUMOURS

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The cavernous sinus syndrome is a well recognised clinical entity. The classical features are ptosis, convergent strabismus, diplopia, intense trigeminal neuralgia and later anaesthesia from pressure on the ophthalmic and maxillary divisions of the trigeminal nerve. Oedema of the eyelids and cornea, exophthalmos, and swelling of the veins of the face from obstruction to the return of venous blood in the sinus may also occur. Dilated retinal veins and papilloedema sometimes are present with marked diminution of vision or blindness, but the disc may be normal and the vision may be only little impaired.

The commonest cause of this syndrome is a thrombosis, usually a septic one but very rarely may be aseptic. The uncommon causes are intracranial carotid and other aneurysms, pituitary tumours, supraseller tumours, meningioma of the lesser wing of the sphenoid, intracranial extension of the nasopharyngeal sarcomas, tumours of the gasserian ganglion, sarcoma of the sphenoid bone, etc.

Association of the cavernous sinus syndrome with a pituitary tumour was first recognised by Foix (1922). Pituitary tumours are by vast majority benign adenomas and they do not produce cavernous sinus syndrome. Jefferson (1940) described a group of uncommon pituitary tumours as 'malignant adenomas' characterised by longer duration than the rare but true malignant tumour, i.e., an adenocarcinoma of the pituitary. They occur usually both in the young and older age groups, and they are rapidly growing extensive adenomas with a tendency of breaking down of the tumour capsule and invasion of the adjacent intracranial structures, most typically involving the cavernous sinus and Meckel's cave. Metastasis does not occur in these tumours and they are practically always composed of chromophobe cells with their nuclei of variable size and density of chromatin net, with numerous mitosis. They constitute about 3 per cent of all cases of adenomas (Henderson, 1939).

Pituitary adenomas usually grow upwards pushing the less resistant thin sellar diaphragm and extend into the cranial chamber. The cancellous bone being less resistant to pressure, excavation of the door of the sella backwards is quite common. But dural and ligamentous tissue forming the lateral wall of the sella are not destroyed by pressure of the benign adenomas. The tumour capsule remaining intact and the structures of the cavernous sinus being sufficiently elastic to adapt themselves to pressure, cavernous sinus syndrome does not occur in such pituitary adenomas. The malignant adenomas in contrast burst through their capsules and the lateral dural and ligamentous walls, and invade the cavernous sinus causing involvement of the oculomotor and trigeminal nerves, and lastly a full picture of the cavernous sinus syndrome. The pituitary gland is surround-

ed by the chiasma and the brain tissue which are remarkably resistant to infiltration except by a tumour derived from their own tissue. Hence extension into the cavernous sinus and Meckel's cave are the only signs of malignancy of an adenoma and such invasion envelops nerves and vessels without breaking through their sheaths.

According to Weinberger *et al* (1940), a partial paralysis of the oculomotor nerve is common with ptosis and paralysis of the upward movement of the eye. The trigeminal nerve is affected with pain, paraesthesia, impaired sensation or absent corneal reflex. Proptosis is present in a few cases. Oculomotor and trochlear nerves are more often involved than the abducent nerve due to interposition of the carotid artery.

Average cerebrospinal fluid protein in malignant adenomas was raised to about 130 mg. per cent in Jefferson's series. The skiagram of the skull shows, in most cases of the malignant adenomas, the entire disappearance of the outline of the sella but rarely a normal sella may be seen. Extension into the cavernous sinus may cause erosion and ballooning of one side of the sella only and on a lateral view the sella appears to have a double outline. These malignant adenomas, though not immediately fatal, are prognostically bad. Deep x-ray therapy is of little use and they do very badly with surgical treatment because of high operative mortality (33 per cent in Jefferson's series).

The next common tumour in the pituitary region is craniopharyngioma, arising from the squamous epithelial cell rests, usually along the pituitary stalk and situated above the sella. It may also develop within the sella when it springs from the cell groups dispersed into the anterior lobe of the pituitary gland, and very rarely this tumour may develop within the sphenoid bone. Commonly they are malignant, and usually epidermoid carcinomas but basal cell carcinoma may occur. Benign tumours are not uncommon (Boyd, 1950). They are usually cystic but may be solid, or partly cystic and partly solid, or even cystic-papillary in structure (Anderson, 1948). Suprasellar craniopharyngiomas are easily diagnosed for their wellknown clinical and radiological features, but intrasellar and intrasphenoidal craniopharyngiomas are often indistinguishable from the pituitary tumours without operation or biopsy (Greene, 1951). Extensions of craniopharyngiomas upwards producing chiasmal and hypothalamic syndromes and downwards producing hypopituitarism are quite common. The intrasellar and intrasphenoidal craniopharyngiomas crowd out the hypophysis causing extreme atrophy, and widening and deepening of the sella. They may completely destroy the sella and even large parts of the sphenoid bone. If the craniopharyngioma is carcinomatous, it infiltrates the adjacent parts of the skull and brain (Anderson, 1948). Extensions into the cavernous sinus producing cavernous sinus syndrome and into the nasopharynx producing nasal obstruction are of extremely rare occurrence; and possible for a carcinomatous intrasellar or intrasphenoidal type of craniopharyngioma. The carcinomatous type of craniopharyngioma occurs more often in young persons, equally in both sexes and may attain a large size.

Recently we have observed two cases of cavernous sinus syndromes due to pituitary tumours, the reports of which are as follows.

Case Report

Case 1—A female aged 21 years was admitted on 25th September 1954 with the history of headache of insidious onset of about 8 months' duration. The headache was felt more over the forehead and gradually became severe and bursting in type. Along with headache she had noticed gradual failure of vision of the right eye. Five months before admission she had complete ptosis of the left side which improved partly on treatment, but she was left with partial ptosis and diplopia. The diplopia persisted till she became blind in her right eye within a month. For last two months she had gradual dimness of vision in her left eye also; and she developed a burning sensation on the left side of the forehead and on the left cheek which became numb. She had complete amenorrhoea for last four years. Her menstruation previously was regular.

On examination she was of average build and showed definite proptosis of the left eye with partial ptosis. There was divergent squint on the left side and restriction of the upward and the inward movements. Both the pupils were dilated and without any reaction to light. Perception of light was lost on the right side and vision on the left side was limited to finger counting at six feet, mostly on the nasal half of the field. There was complete primary optic atrophy on the right, and partial primary optic atrophy of the left. Hypoaesthesia and hypo-algesia was present in the distribution of the left ophthalmic and maxillary divisions of the trigeminal nerve. No other abnormal features were detected.

Wassermann reaction of blood was weakly positive and Kahn test was negative. Skiagram of the skull showed enlargement of the sella turcica with complete destruction of the posterior clinoid processes and the anterior clinoid processes were eroded and sharpened (Fig. 1, *vide* Plate).

Within a fortnight of her admission the proptosis on the left side rapidly increased. The conjunctiva at first was congested, but later marked chemosis of the conjunctiva and oedema of the eyelids developed. Proptosis though very marked was not reducible and not pulsatile. All the movements of her left eye were markedly

restricted. She had severe discomfort in her left eye and she became completely blind. The fundus showed complete primary optic atrophy on the left and slightly congested retinal veins. There was no rise of temperature and no leucocytosis. The patient took her discharge and no follow-up was possible.

Case 2—A young man of 21 years was admitted on 18-1-1955 with the history of painful swelling of both the eyelids and upper part of the face, a discharging sinus near the internal canthus of the left side, and fever of 4 days' duration. Prior to these recent urgent symptoms he was noticing gradual diminution of vision of both the eyes for about a year. Three months back he became completely blind and both the eyes started gradually bulging. Two months back he developed increasing obstruction of the left nostril with occasional profuse epistaxis. He lost good amount of weight and his general condition was poor. About five days before admission he developed a painful swelling near the internal canthus of the left side with fever and swelling of both the eyelids and upper part of the face.

On examination a discharging sinus was present over the left lacrimal sac area with swelling of the upper part of the face. Both the eyes showed marked proptosis and swelling of the eyelids and chemosis of the conjunctivae, more prominent on the left side (Fig. 2, *vide* Plate). Proptosis was not reducible and not pulsatile. Pupils were widely dilated and not reacting to light, light perception was lost on both sides. All external ocular movements were lost and eyes were fixed at the centre. There was complete primary optic atrophy of both the eyes and retinal veins were not congested. Hypo-aesthesia and hypo-algesia were present in the distribution of the left maxillary division of the trigeminal nerve and corneal reflexes were lost too on both sides. There was serosanguinous discharge from the left nostril and marked deviation of the septum to the right and the whole cavity was filled up with a solid mass. Skull on routine examination was normal.

He was poorly nourished and his temperature was varying between 99° and 101°F. His blood examination did not show leucocytosis and he was not toxic. Antibiotic treatment did not improve much of his eye condition, though it brought down the temperature to normal.

Skiagram of the skull showed complete destruction of the sella with a large part of the sphenoid bone, and marked widening of the nasal cavity with deviation of the nasal septum to the right (Figs. 3 and 4, *vide* Plate).

A biopsy was done through the left nostril to remove a piece from the solid mass. The tissue thus removed, on histological examination, showed marked anaplastic type of carcinoma cells in a craniopharyngioma, i.e., a malignant craniopharyngioma (Fig. 5, *vide* Plate).

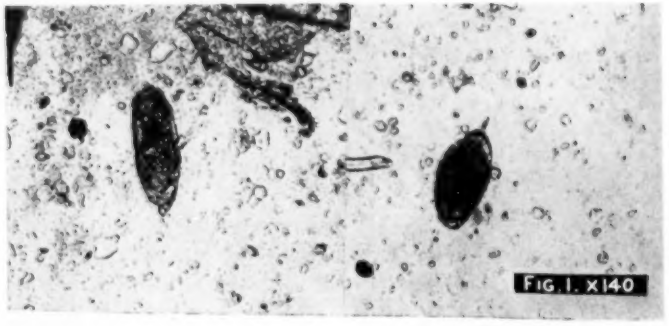
Within a week of his admission the proptosis of the left eye markedly progressed and the whole eyeball was pushed forwards and laterally; and a firm round swelling appeared at the medial side of the upper eyelid which later on broke down to a large round solid growth (Fig. 6, *vide* Plate). Later on there was marked deterioration of the general condition of the patient and he took his discharge against advice.

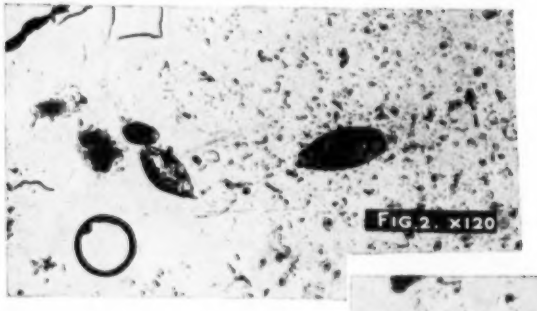
Comments

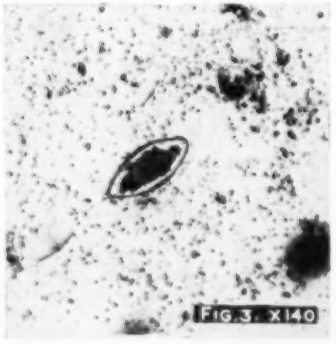
Case 1 first consulted an ophthalmologist with dimness of vision in the right eye and complete ptosis of the left eye. Ptosis may be the first presenting symptom of a pituitary adenoma as noted by Hirsch (1921). Partial improvement of ptosis suggested that it might be due to simple lateral pressure of the tumour before its invasion through the medial wall of the cavernous sinus. The associated diplopia was not an indication of extension, as Jefferson noted it to be a common symptom due to simple pressure. In the hospital she presented herself with features of predominant uniocular involvement, as manifested by blindness of the right eye and partial temporal field defect of the left eye ; which suggested a lesion at the anterior end of the chiasma mainly at the base of the right optic nerve. According to Jefferson this type of lesion can be produced by localised herniation of an adenoma through the capsule impinging on the base of one optic nerve. The gradual deterioration of the left eye was possibly due to more complete pressure on the middle of the chiasma involving the fibres of both the eyes. This delayed onset of the bilateral field defect in presence of evidences of extensions could be due to associated post fixation of the chiasma. The presence of proptosis, partial oculomotor palsy, and trigeminal anaesthesia on the left side was sufficient evidence to diagnose this case as 'malignant adenoma'. In the later stages she developed almost a complete picture of the cavernous sinus syndrome. History of amenorrhoea for 4 years suggested that the tumour probably started 4 years ago.

Case 2 had the presenting features of cavernous sinus thrombosis ; but the absence of marked toxaemia, high fever, significant leucocytosis, response to antibiotics ; and the presence of optic atrophy on both sides made the diagnosis untenable. On further inquiry the sequence of events in the history were : blindness of both eyes for 3 months, nasal obstruction and epistaxis for 2 months, the marked proptosis with ophthalmoplegia and the subsequent course as described, all suggested a tumour in the pituitary region. But a nasopharyngeal sarcoma growing simultaneously upwards invading the cranium and downwards producing nasal obstruction and epistaxis could not be ruled out clinically. Skiagram did not help at all at this crucial point. The biopsy from the nasal mass gave the final diagnosis.

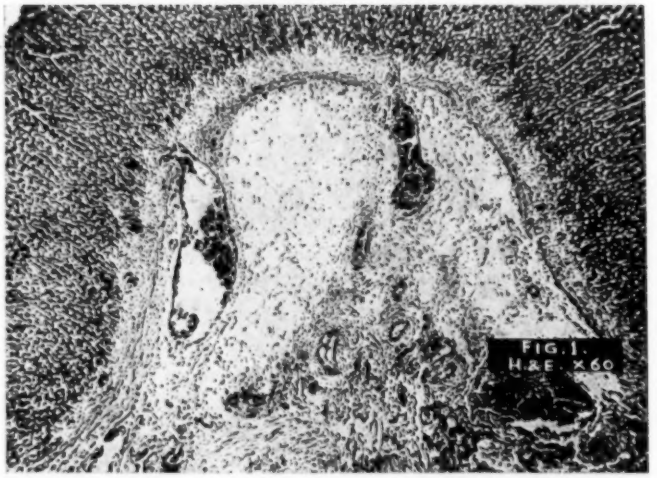
As the case presented at a very late stage it was not possible to diagnose definitely the type of the craniopharyngioma. But the history of blindness for a long time, optic atrophy, complete destruc-



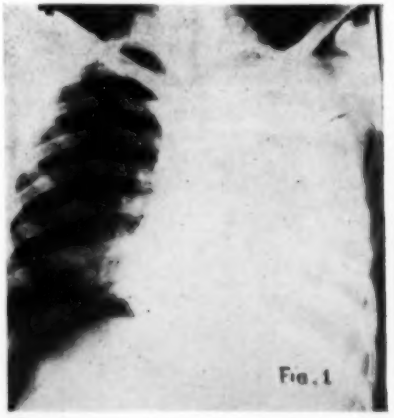


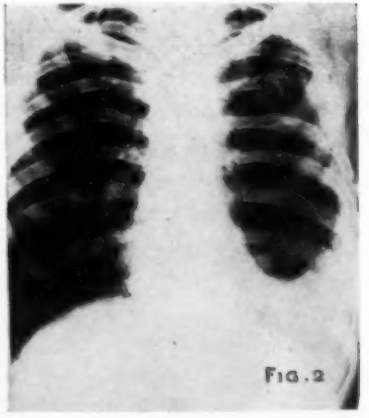


DHANDA—*Infestation with Ova Morphologically Resembling Schistosoma Haematobium* (*pp. 407-08*)



BASU MALLIK—*Histamine Shock in Adrenalectomised Rats* (*pp. 408-10*)





KARAI—*Decortication in Clotted Haemothorax* (*pp. 410-12*)

Fig. 1—Enlargement of the sella turcica with complete destruction of the posterior clinoids and erosion and sharpening of the anterior clinoids.

Fig. 2—Proptosis and swelling of the eyelids on both sides with a discharging sinus over the left lacrimal sac area.

Fig. 3—Complete destruction of the sella turcica with a large part of the sphenoid bone.

Fig. 4—Marked bony destruction with widening of the nasal cavity and deviation of the nasal septum to the right.

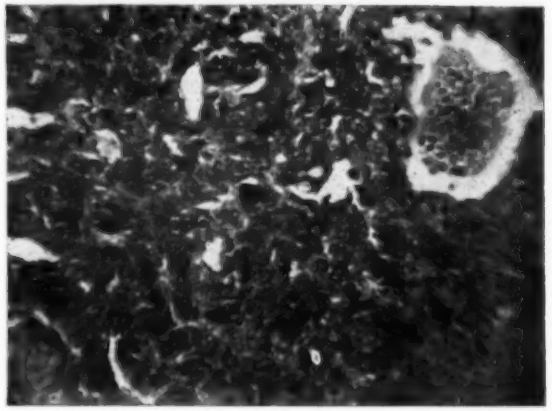


Fig. 5—Photomicrograph illustrates anaplastic type of cancer cells around a vessel. (H. & E. × 225)

Fig. 6—The left eyeball showing marked proptosis and pushed laterally by a swelling at the medial side of the upper eyelid.

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tion of the sella, and nasal obstruction by the same growth suggested a possibility of the intrasellar or intrasphenoidal type of craniopharyngioma. Further differentiation between the intrasellar and intrasphenoidal types was not possible. The infiltrating nature of the carcinomatous type of craniopharyngioma into the adjacent parts of the skull and the intracranial structures was possibly the reason of production of the cavernous sinus syndrome in this case. The young age of the patient and the large size of the tumour, as evidenced from the skiagram, were points in favour of a malignant craniopharyngioma, but no doubt the biopsy gave the final answer to it. The acute dacryocystitis with discharging sinus, which brought the patient to the hospital, was a secondary septic complication due to nasal duct obstruction from the tumour.

Summary

Features of the cavernous sinus syndrome in pituitary tumours have been briefly described with a short review of the literature. Two cases have been reported, one of which was a malignant pituitary adenoma and the other was a malignant craniopharyngioma.

Acknowledgments

Our grateful thanks are due to Dr. M. N. Sarkar, Superintendent, Chittaranjan Hospital for his permission to publish these cases. Our hearty thanks to Dr. A. C. Guha, Professor of Pathology, and to the staff of his department for kindly supplying us with the microphotograph. We also wish to thank Dr. J Ghosh and Dr. S. Bhakta for their kind co-operation all through.

References

Anderson, W. A. D.—Editor, Pathology, First Edition, 1948. C. V. Mosby Company, St. Louis.

Boyd, W.—The Pathology of Internal Diseases, Fifth Edition, 1950. Henry Kimpton, London.

Duke-Elder, Stewart—Text Book of Ophthalmology, Vol. IV, 1949, C. V. Mosby Company, St. Louis & Henry Kimpton, London.

Foix (1922)—Quoted from Greene, Raymond (1951).

Greene, Raymond—The Practice of Endocrinology, Second Edition, 1951, Eyre & Spottiswood Ltd., London.

Henderson, W. R.—*Brit. J. Surg.*, **26** : 811, 1939.

Hirsch (1921)—Quoted from Duke-Elder, Stewart (1949).

Jefferson, G.—*Proc. Roy. Soc. Med.*, **33** : 433, 1940.

Weinberger, L. M., Adler, F. R. and Grant, P. C.—*Arch. Ophthalmol.*, **24** : 1197, 1940.

PRACTITIONERS' SERIES

PERICARDITIS

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Pericarditis is not an uncommon disease. Quite often the diagnosis is missed because of the examiner not being aware of its frequency and its clinical picture.

The various clinical types are the acute dry pericarditis, pericarditis with effusion, constrictive pericarditis and adhesive pericarditis (pericardio-mediastinitis).

Aetiologically, it may be tuberculous, rheumatic, pyogenic, uraemic, malignant or traumatic in origin. It may be also secondary to myocardial infarction or may be the so-called benign variety.

Dry pericarditis—The onset of the dry pericarditis may be asymptomatic or may be accompanied by discomfort and pain in the precordium and depending on the cause, malaise and fever may be present or if already present, may increase. The only physical sign of dry pericarditis is the pericardial rub which may be audible all over the precordium or may be localised. If localised, usually it is best marked in the left 4th intercostal space in the parasternal line. Sometimes the to-and-fro rub may be audible only when the patient is leaning forwards and the breath held after a forced expiration. Usually the manoeuvre makes the rub more prominent. Sometimes a rub may be palpable.

Pericarditis with effusion—Formation of effusion may further increase the toxaemic symptoms and discomfort in the chest but may remain asymptomatic.

The physical signs of pericarditis with effusion are as follows:

Bulging of the precordium due to rheumatic pericarditis is rarely seen except in children. The cardiac impulse remains palpable in half the cases inspite of the effusion and since it is elicitable in about the same proportion of normal healthy adults only its disappearance can have any value. Presence of dullness beyond the apex beat is a reliable sign. Cardiac dullness may extend upwards to the second or first intercostal space in the left parasternal line and when the impairment becomes less or disappears on the patient's sitting up from the lying position, it is diagnostic of pericarditis with effusion. This shifting dullness is present in the majority of patients with pericardial effusion. The postural change

may be absent when there is a tense massive effusion or the sign may be vitiated if there is consolidation of the upper lobe of the left lung.

Heart sounds may be normal or distant. Pericardial rub is present in half the cases of pericarditis with effusion and again may be best elicited with the patient leaning forwards after a forced expiration. Apart from those cases in which there is concomitant valvular disease usually rheumatic, there is as a rule no murmur. Occasionally a mitral soft systolic murmur may be present. The lungs unless involved also in the causative disease process, e.g., tuberculosis, do not show any constant physical signs. In about one third of the cases, there may be signs of partial collapse consolidation in the lower lobe of the left lung due to massive effusion. Fine crepitations in left infrascapular region with or without diminished air entry are present. Rarely the percussion note may be impaired in the same region.

If the effusion has formed rapidly or is large enough, signs of cardiac tamponade may be present. They depend upon rise of intrapericardial pressure so that the right auricular pressure is approached and venous inflow is impeded. Neck veins become full and venous pulsation diminishes. Jugular venous pressure is raised above the sternal angle. Liver becomes enlarged and tender and there may be ascites and later on oedema of feet. The whole picture simulates right heart failure and is often missed as such. But in contrast to right heart failure the dyspnoea is much less, usually present on exertion only, the fullness of the neck veins changes little with change of posture, the venous pulsation is diminished, oedema of feet appears later and is less marked than ascites.

In cardiac tamponade, due to diminished venous return, cardiac output is diminished and hence the blood pressure falls. The systolic blood pressure is usually less than 110 mm. Hg. Pulse rate is increased usually to above 80 per minute and the volume is low. The extremities are pale and cold.

Constrictive pericarditis—The symptoms and signs of constrictive pericarditis are similar to those of pericarditis with effusion causing cardiac tamponade. Fibrous encasing over the visceral layer of the pericardium prevents diastolic stretch of the right auricle and causes obstruction to venous inflow. The patient comes with a history of dyspnoea on accustomed exertion but there is seldom any dyspnoea at rest. As the usual cause is a previous tuberculous pericarditis, there may be pulmonary and pleural manifestation of haematogenous spread of tuberculosis and the patient may have low grade fever and cough which may be productive. The patient may also complain of swelling of the abdomen and feet. Examination shows evidence of recent loss of flesh, cold hands and a rapid small volume pulse. The venous pressure is raised, the neck veins full, liver is enlarged and tender and there is usually ascites. There may or may not be oedema of the feet. The cardiac impulse may be visible. The heart size as judged from the position of the apex beat may be normal or slightly enlarged. There is no dull percussion note extending beyond the apex beat and no shifting dullness in the left second space. The heart sounds are normal. Rarely there may be soft systolic mitral murmur. The cardiac rhythm remains unaltered. Rarely, there may be auricular fibrillation. The differentiation from right-sided cardiac failure is made on the same criteria as in the case of pericarditis with effusion causing cardiac tamponade. The lungs may be clear or may show signs due to associated pulmonary tuberculosis or pleural effusion which may be the result of back pressure or due to tuberculous pleurisy.

Adhesive pericarditis—Adhesive pericarditis is a less common clinical entity. It is usually the result of previous rheumatic pericarditis. It produces no symptoms as such. The physical signs attributed to it are due to the associated chronic mediastinitis causing adhesions between the pericardium and the structures round the mediastinum fixing the heart to the surrounding bony cage. Thus we may have the fixation of the apex beat which does not change its position with moving the patient from side to side or retraction of the lower part of sternum or lower left ribs at the back with each cardiac systole. The heart is usually enlarged and there may be murmurs due to concomitant rheumatic valvular disease.

Radiologically pericarditis with effusion shows an enlarged heart which is triangular in shape with the left border straightened. If the effusion is massive the heart may take a globular shape. The cardiohepatic angle remains acute both in the postero-anterior and lateral views. Fluoroscopic examination shows very poor or absent cardiac pulsation. The lung fields are usually clear and this contrasts with dilatation of the heart secondary to failure.

In constrictive pericarditis the changes are similar except that the heart is not enlarged or only moderately enlarged.

Electrocardiogram may help in the diagnosis.

The diagnosis of pericardial effusion may be confirmed by pericardial paracentesis.

The aetiological diagnosis can be made by proper assessment of history, physical signs in

the heart and elsewhere and course of the disease including its response to treatment.

Rheumatic pericarditis usually occurs in the young, there is usually a history and signs of subacute rheumatic fever including anaemia, sorethroat, joint pains and swellings, subcutaneous nodules, chorea and cardiac murmurs. The effusion is seldom massive. It is usually serous but sometimes it may be haemorrhagic. Response to adequate salicylate therapy is dramatic. It may cause adhesive pericarditis or pericardiomediastinitis but does not lead to constrictive pericarditis.

Pyogenic pericarditis may be the result of spread from bacterial inflammation of the lung or pleura or part of a pyaemic process. In both, the patient is usually severely ill, signs of cardiac tamponade appear early and may require repeated aspiration with intrapericardial instillation of penicillin. In pneumococcal pericarditis the pus may be very thick and may require open drainage though early diagnosis and treatment may help to avoid that. Systematic treatment with a suitable antibiotics will also be necessary. The pericardial exudate is rich in polymorphs.

Pericarditis following myocardial infarction is usually dry. The rub appears a day or two after the infarction and the history makes the diagnosis clear.

Tuberculosis is the commonest cause of pericarditis in our country and like idiopathic pleurisy involves chiefly young adults. It may be the result of direct spread from the mediastinal glands affected in the primary complex but is more often a manifestation of haematogenous spread and may be accompanied or followed by tuberculosis in other parts of the body. Often, it may be part of polyserositis. The onset, as in most cases of tuberculosis elsewhere, is often insidious and there is malaise, fever and loss of weight. Raised E.S.R. may not be very marked. Cough, breathlessness on accustomed exertion, fever, swellings of legs and abdomen are the common symptoms.

Though it may resolve completely after the dry stage, the patient usually has signs of cardiac tamponade due to effusion by the time he comes to the doctor. Oedema of the ankles, enlarged firm liver, raised pulse rate, ascites, raised jugular venous pressure and low systolic blood pressure, shifting dullness in the left second space and pericardial rub were the physical signs in this order of frequency in a series studied by the present writer. The effusion may be haemorrhagic. In some of these cases, the exudate may be too thick to aspirate. Such cases, classified as subacute tuberculous pericarditis simulate very much constrictive pericarditis. Only the follow up when in response to adequate anti-tuberculous chemotherapy or even without, signs of cardiac tamponade diminish and the heart regains normal size, clinches the diagnosis.

Sometimes, it is possible to isolate tubercle bacilli on culture or guineapig inoculation of the pericardial fluid. But more ofter the diagnosis with regard to the tuberculous nature of the effusion has to be made on the history or subsequent development of tuberculous lesions elsewhere in the body, presence or development of signs of inflammation of other serous membranes—pleurae or peritoneum, nature of the pericardial effusion and response to antituberculous chemotherapy.

The tuberculous pericardial exudate may get absorbed spontaneously or with adequate treatment but if it does not resolve, it may lead to constrictive pericarditis. Hence it will be advisable to treat every case of tuberculous pericarditis vigorously on antituberculous lines and start the treatment as early as possible. Besides bed rest and high calorie, high protein, high vitamin diet, antituberculous chemotherapy should be given in each case. Patient may be put on daily streptomycin 1 g. and isoniazid 100 mg. b.d. This may be continued for a period of three months. Pericardial paracentesis may be necessary to diminish cardiac tamponade.

Recently a type of pericarditis has been described mostly in children and young adults which follows a few weeks after an upper respiratory infection and resolves completely within one to three months. It has been variously called benign, non-specific and idiopathic pericarditis. The diagnosis can be made only by exclusion or on follow up. Whether it is a distinct entity due to a definite cause like virus infection or includes mild cases of pyogenic and tuberculous variety which resolve spontaneously, is not decided.

Pericardial paracentesis—The procedure is not difficult. Though it may be done just lateral to the sternum in the 4th left space but avoiding the internal mammary artery or just lateral to the apex beat but inner to the outer border of the cardiac dullness and even from the back, paracentesis from in between the angle between the xiphisternum and the left costal border is probably the easiest and most likely to be successful. The patient is propped up and given a preliminary sedative (morphia ⅓ gr. and atropine sulphate 1/100 gr. by hypodermic injection). The area after proper sterilization is anaesthetised by 2 per cent procaine. A blunt lumbar puncture needle is used. It is introduced in the angle and passed upwards and backwards and laterally and after overcoming the resistance of the diaphragm, it can be felt penetrating the parietal pericardium in a case of

pericarditis with effusion when the fluid begins to drop from the needle. If the needle enters the ventricular muscle it begins to move up and down with each cardiac systole or if it has penetrated deeper, frank blood begins to drip and in both cases the needle should be withdrawn.

In cases of pyogenic pericarditis the pus may be very thick and a wide-bore needle may be necessary. Sometimes a haemorrhagic effusion may look like blood but centrifuging the effusion will show the yellow coloured supernatant exudate in the case of effusion and its haemoglobin estimation in a haemoglobinometer will show seldom more than 10-20 per cent which will be much lower than the patient's haemoglobin. Pericardial paracentesis is not without danger. Rarely there may be sudden collapse or circulatory failure during or soon after pericardial paracentesis. Administration of 0·5 g. to 1 g. of procainamide orally ½ to 1 hour before the paracentesis may minimise the risk involved.

In the end, it may be stressed again that pericarditis is a disease which if looked for, will be found to be more common than most of us have believed hitherto. The search is worthwhile since early diagnosis and consequent early treatment may be able to cure and prevent the development of constrictive pericarditis.

SPECIAL ARTICLE

EMPHYSEMA AND ITS PROBLEMS†

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The problem of chronic hypertrophic emphysema is complex and its treatment difficult.

Emphysema can be conveniently discussed under three heads:

(1) Structural and functional changes.
(2) Anoxia and its consequences.
(3) The treatment of emphysema and its complications.

Structural and Functional Changes

The gross morbid anatomy is wellknown, but certain facts require special mention. Emphysematous bullae and blebs form mainly on the surface of the lung and along its free margins. The changes in deeper tissue are not so severe. Histologically it appears that the large spaces so characteristic of emphysematous lung are formed by rupture of interalveolar septa. But this belief has been challenged by Boyd (1946), who thinks that these spaces may be formed simply by overdistension of alveoli, and what appear to be ruptured septa are in fact intact septa widely separated by overdistension of the spaces. The whole lobular structure is also distorted and distended and the alveolar duct is funnel-shaped.

The pulmonary capillaries are narrow and in places completely obliterated. Cudkowicz and Armstrong (1953) showed two main types of changes in bronchial arteries—(1) complete occlusion of bronchial arteries and (2) an anastomosis between bronchial and pulmonary arteries. While in the former ischaemic changes of structures supplied by the bronchial artery is quite evident, changes in connective tissues of the lung in the latter are explained by them as the result of diversion of bronchial artery blood into the pulmonary artery circulation. Ischaemia thus produced may be responsible for the destruction of interalveolar and peribronchial connective tissue. Abbot *et al* (1953) think that the ischaemia is produced by reflex arteriolar spasm. They argue, as partial occlusion of the femoral vein causes reflex spasm of femoral artery, a similar pathological condition may produce bronchospasm and consequently interfere with the pulmonary artery circulation causing reflex spasm of bronchial artery system. If the condition is chronic, ischaemic changes occur in the connective tissues of lungs. They think that any local pathology like bronchiectasis, or a generalised bronchospasm may bring about this circulatory change. If any of these explanations is correct the rupture of alveolar walls and formation of blebs and bullae, specially near the surface, can be attributed to the peripheral ischaemia. The pull of intrapleural negative pressure helps in the process.

The aetiological association of bronchial asthma and emphysema is generally thought to be consequential and the alveolar distension is regarded as the result of obstruction to the expiratory air due to bronchoconstriction. It is also a common belief that occupations involving great expiratory effort like those of glassblowers and trumpet players are associated with emphysema of lungs, though Christie (1939) could not find any such aetiological relation. If expiratory obstruction is a significant causative factor, the lungs should be distended uniformly. Moreover, during expiration the lung surface is supported by the chest wall; this should prevent overdistension near the surface and the deeper tissues should suffer more.

† Based on a peper read at the Calcutta Medical College Reunion (1954-55).

Some authorities try to view the problem from the opposite angle. They believe that forced inspiratory efforts may be responsible for overdistension of the alveoli and weakening of their walls. Just before coughing or forced blowing there is a sudden deep inspiration. Chronic cough is a constant companion of patients suffering from chronic bronchitis and asthma, and repeated forced inspiration is a necessary evil in occupations involving blowing. This may explain emphysema of chronic bronchitis and asthma, even if there is no occupational emphysema.

Christie (1952) reviewed these hypotheses and explained the mechanism by the theory of repeated alveolar stress and strain. This is a sort of synthesis of expiratory and inspiratory theories. During the initial phase of coughing a deep inspiration is followed by closure of the glottis and the lungs are compressed by contraction of muscles of the chest and abdominal walls and rise of diaphragm. In consequence there is a great rise of intra-alveolar pressure (Kountz and Alexander, 1934 ; Rasmussen and Adams, 1942). This is followed by sudden release of pressure when the glottis opens and the trapped air is expelled forcefully. This cycle is then repeated. This constant strain of repeated pressure changes, the magnitude of which is very great indeed, compared to the delicate structure of lung alveoli, may be a reasonable explanation if it is considered how frequently a history of chronic cough, whatever its aetiology, is associated with the developmental time table of chronic hypertrophic emphysema.

That there is loss of elasticity of the lung is beyond doubt. This is not only seen post-mortem but also in life. Christie (1934) has shown that the intrapleural negative pressure is greatly reduced, approaching atmospheric pressure, even in deep inspiration. What is not very clear is how it is brought about.

McIlroy and Christie (1952) experimentally studied the elastic property of lung post-mortem and found that the organ is in fact visco-elastic, i.e., an organ whose elastic property is a combination of a viscous and an elastic system. They defined viscous system as one in which the response to a force depends on the time when it is applied and an elastic system as one in which the response depends on the magnitude of the force applied. The elasticity of such an organ may therefore be reduced either by an increase of viscous element, or by loss of elastic element. If there is increase in viscous element the lung will take longer time to respond to changes of pressure of inflation and deflation, so that both the phases of respiration are cut short. If there is absolute loss of elastic element there is increased distensibility of the organ to a given pressure, there being no elastic recoil to counteract it. The volume of lung increases till it is limited by the rigid thoracic wall.

McIlroy and Christie (1952) think that the dyspnoea on exertion at old age is due to increase in viscous element, i.e., due to relative loss of elasticity. In emphysema, on the other hand, there is absolute loss of elasticity as is shown by reduction of intrapleural negative pressure. The distensibility of lung is increased and the increased volume of lung is accommodated inside a chest which assumes a fixed inspiratory position. There is evidence that the viscous element of lung is also increased. The maximum breathing capacity is reduced possibly because a more viscous organ is unable to respond readily to increased rate and depth of respiration. The tendency of a more viscous organ to lag behind the chest wall during inspiratory change of intrapleural pressure may be responsible for the formation of blebs and bullae, specially on free surface and margins of the lung. The ischaemia of peripheral alveoli may help in the process. The characteristic curve of vital capacity tracing in emphysema may also be due to increased viscosity of lung.

This work is undoubtedly a step forward, but what is actually responsible for the increased viscous property is not known. It may be that some people are born with more viscous lungs and are predisposed to develop emphysema. This, however, does not explain the increased viscosity in old age.

Anoxia and Consequences

In emphysema total lung capacity, residual air and functional residual air are increased ; vital capacity and maximum breathing capacity are reduced ; and intrapulmonary mixing of gases is much delayed. These data of pulmonary function are very important for diagnosis, prognosis and pre-operative assessment. The main baffling problem is, however, one, i.e., anoxia. Oxygen saturation of arterial blood is reduced and there is retention of CO_2. Alkali reserve is consequently increased.

Various factors have been considered as responsible for the production of anoxia. The impaired inspiratory expansion of chest may be responsible for impaired ventilation. The impaired function of inspiratory muscles is a mechanical disadvantage due to fixation of chest wall in a position of inspiration. But emphysema and anoxia may be present even without this extreme anatomical change and anoxia may be present when the amount of air breathed per breath and per minute is greater than normal (Christie, 1952).

It has already been observed that there is dilatation and distortion of the alveolar duct. This may mean that a jet effect of air current in the alveolus necessary for normal ventilation is lost. But physiologists must agree that normally ventilation occurs by a jet of air impinging on alveolar epithelia and not by diffusion.

The hypothesis of a functional barrier to exchange of gases at alveolar epithelium seems most unlikely as the changes in gaseous saturation in the alveoli and blood are in equilibrating proportion (Christie, 1952). Destruction of capillary bed, however, may be partly responsible for impairment of haemorespiratory function.

The increase in anatomical dead space due to dilatation and distortion of bronchioles is too small to be of great import (Hoover and Gammon, 1915). In a normal individual distribution of ventilation is remarkably uniform throughout the lung. But in emphysema samples of expiratory air contain increasing amount of CO_2 with increased depth of expiration. There is also a great delay in intrapulmonary mixing of gases in emphysematous lungs (Cournaud *et al*, 1941 ; Bates and Christie, 1950). These facts suggest that in emphysema a pathological dead space is formed at the periphery of the lungs. Anatomical evidence of such dead space is found in the blebs and bullae. It is quite reasonable to expect that a large amount of inspired air is diverted to the avascular blebs and bullae which are unnecessarily overventilated at the expense of relatively healthy and vascular parts of the lung. Finally, bronchial asthma and other primary aetiological factors may themselves interfere with normal ventilation and thus contribute towards anoxia.

Anoxia of emphysema creates certain respiratory, circulatory and neurological problems, which the clinicians are confronted with. Patient is already handicapped with anoxia and any increased demand for oxygen is returned with a note of bankruptcy. Unproductive respiratory effort for more oxygen not only exhausts the patient but causes a further increase in demand and a vicious circle is established. This is evident when he undertakes some physical exertion or when the ventilatory efficiency is further reduced by some pulmonary infection.

More intricate is the problem of circulatory changes and heart failure. The anoxia produces a hyperdynamic circulatory state and even in heart failure the output remains high normal or even above normal. The only exception to this is found when the systemic blood pressure falls, and the fall in output is then proportional to the fall in systemic blood pressure (Howerth *et al*, 1947 ; Richards, 1947). The raised venous pressure in this high output failure is considered to be a compensatory mechanism, an active attempt to keep up adequate venous filing (McMichael, 1946) for the increased circulatory need.

The functioning lung is essentially a capillary organ and probably about 50 per cent of pulmonary blood is accommodated in the capillary bed (Daley, quoted by McMichael, 1952). Consequently an enormous area of capillary bed has to be eliminated to produce pulmonary hypertension of sufficient degree. The cardiac disability is not, by any means, proportional to the degree of emphysema. Furthermore, with the progress of thoracic surgery convincing proof has accumulated regarding the amazing power of the human system to adjust to the respiratory and circulatory need of the body. A properly assessed subject does not develop right ventricular failure after removal of an entire lung.

In a normal person at rest, the systolic and diastolic pressures in the pulmonary artery are between the ranges of 18 and 30 mm. and 8 and 10 mm. of Hg. respectively, as measured directly by catheter. With exercise the systolic pressure remains unaltered and the diastolic pressure falls. Riley *et al* (1946) think that this is brought about by a reflex vasodilatation, so that the right ventricle is spared of undue strain of increased output in response to increased circulatory demand. This physiological mechanism fails in emphysema. Here the pulmonary artery pressure at rest is either normal or higher than normal (Riley *et al*, 1946) ; and with exercise both systolic and diastolic pressures rise steeply. This rise of pulmonary artery pressure is also seen at rest when there is a superadded respiratory infection (Bloomsfield *et al*, 1946 ; Lenegre and Maurice, 1948). Motley *et al* (1947) observed that in normal individuals breathing in an atmosphere containing 50 per cent of normal concentration of oxygen causes a marked rise in the pulmonary artery pressure. Anoxia of emphysema, specially under aggravating condition like physical exercise and respiratory infection may similarly cause a rise in pulmonary artery pressure. This rise has been explained by inhibition of reflex arteriolar dilatation and simultaneous increase in cardiac output. In the author's experience there is a definite reduction in venous pressure with administration of oxygen alone. Mounsey and his colleagues (1952), however, failed to reduce pulmonary artery pressure by such means. They found that slight but definite increase of haemoglobin concentration was associated with right heart failure. The matter is still *sub judice*, but it is almost definite that anoxia plays a great part in the production of pulmonary hypertension.

Treatment of Emphysema and Its Complications

With these facts in view the difficulty of management of such a case can be easily appreciated. Chronic hypertrophic emphysema is not only an irreversible process but also a condition the development of which can hardly be prevented. The treatment consists mainly of treatment of its complications. The unfortunate subject is rendered completely crippled, incapable of any physical exertion and is an easy prey to respiratory infection.

During an acute respiratory infection patient develops clinical features of increased intracranial tension and papilloedema (Simpson, 1948; Cameron, 1933). Again Davis and MacKinnon (1949) and Westlake and Kaye (1954) showed that administration of oxygen to anoxic emphysematous patients causes a steep rise in C.S.F. pressure and this is associated with certain mental and neurological symptoms like acute mania, disorientation, sweating, salivation, myoclonic twitching, stupor and coma. The rise of C.S.F. tension appears to be due to altered haemodynamic state due to retention of CO_2. Carbon dioxide causes dilatation of cerebral arteries (Kety and Schmidt, 1948; Patterson *et al*, 1952) and the increased cerebral circulation causes increased production of C.S.F. Associated increase in venous pressure in cor pulmonale may retard absorption and cause further rise in C.S.F. tension (Donald, 1949). The question is whether the symptoms are due to increased intracranial tension. Westlake and Kaye (1954) argued that drowsiness and coma of intracranial tumours which are also associated with high C.S.F. tension, are due to decreased blood flow and consequent cerebral anaemia. But in the condition under discussion there is increased cerebral blood flow. It seems that the mental and neurological symptoms are symptoms of CO_2 intoxication and has nothing to do with increased intracranial tension. Lumbar puncture does not improve these symptoms.

It appears anomalous how improvement of anoxic state with administration of O_2 can be associated with intoxication due to CO_2 retention. In emphysema there is already a high CO_2 content in blood and the respiratory centre gets refractory to the stimulating effect of CO_2. The patient's respiratory activity depends on anoxic stimulation of aortic and carotid sinuses. Administration of oxygen relieves the anoxia and removes this stimulus. Resulting hypoventilation causes further retention of CO_2. Such hyperapnoea invariably occurs during oxygen therapy in emphysematous subjects and perhaps many deaths have occurred as a result of CO_2 intoxication by injudicious methods of oxygen therapy. The more severe the anoxia the more is the possibility of untoward symptoms (Richards, 1950). At the same time, the more severe the anoxia the more urgent is the need for oxygen therapy. Barach (1948) recommends administration of oxygen by nasal catheter at the rate of 3 litres per minute to start with. It can then be gradually worked up, or, as an alternative, when the patient is acclimatised to a high concentration of oxygen he may be transferred into a tent. He thinks that patients tolerate a high arterial CO_2 if it is gradually produced (Barach, 1950).

One of the common modes of fatal terminations is right heart failure, which is usually precipitated by respiratory infection. While adequate treatment of infection with suitable antibiotic and of anoxia with oxygen cannot be too strongly emphasised, the treatment of congestive failure *per se* is a great problem. As the increased venous pressure is regarded as a compensatory mechanism for the maintenance of increased cardiac output, venesection is thought to be absolutely contra-indicated. Howarth *et al* (1947) would not even recommend digitalis, because they think that digitalis acts primarily by reduction of venous pressure and thereby venous filling. The only permissible indication, according to them, is when the systolic blood pressure falls below 100 mm. Hg, or in other words when the failure turns into a low output failure. But at this stage there is hardly any chance of getting the patient out of the woods.

Mercurial diuretics also lower the systemic venous pressure, though the mechanism may not be the same as digitalis. Their use, however, is not fraught with danger under such circumstances. On the contrary, they may be very useful. It is doubtful if digitalis acts primarily by lowering venous pressure. It seems that it acts essentially by improving cardiac action. Although the effect of digitalis in these cases is unpredictable, it is no longer regarded as an absolute contra-indication. Mounsey *et al* (1952) found immediate increase of right ventricular pulse pressure with digitalis therapy. Aminophylline definitely lowers the venous pressure and acts well in cor pulmonale.

References

Abbot, O. A., Hopkins, W. A., Van Fleit, W. E. and Robinson, J. S.—*Thorax*, **8**: 116, 1953.

Barach, A. L.—Physiologic Therapy in Respiratory Diseases, 2nd Edition, 1948, Lippincott, Philadelphia.

Idem—*J. A. M. A.*, **143**: 1047, 1950.

BATES, D. V. AND CHRISTIE, R. V.—*Clin. Sc.*, **9**: 17, 1950.

BLOOMFIELD, R. A., LAUSON, L. D., COURNAUD, A., BREED, E. S. AND RICHARDS, D. W.—*J. Clin. Invest.*, **25**: 637, 1946.

BOYD, W.—Pathology of Internal Diseases, 4th Edition, 1946, Published by Lea & Febiger, p. 223.

CAMERON, A. J.—*Brit. J. Ophth.*, **17**: 167, 1933.

CHRISTIE, R. V.—*Edinburgh M. J.*, **46**: 463, 1939.

Idem—Diseases of Chest, Vol. 2, Edited by Marshall, G. and Perry, K. M. A., 1st Edition, 1952, Butterworths Ltd., London, p. 115.

Idem—*J. Clin. Invest.*, **13**: 295, 1934.

COURNAUD, A., BALDWIN, E. D., DARLING, R. C. AND RICHARDS, D. W.—*J. Clin. Invest.*, **20**: 681, 1941.

CUDKOWICZ, L. AND ARMSTRONG, J. B.—*Thorax*, **8**: 46, 1953.

DALY, I. DEBURGH—Quoted by McMichael (1952).

DAVIS, C. E. AND MACKINNON, J.—*Lancet*, **2**: 883, 1949.

DONALD, K.—*Ibid*, **2**: 1056, 1949.

HOOVER, C. F. AND GAMMON, J. E.—*Arch. Int. Med.*, **15**: 501, 1915.

HOWARTH, S., MCMICHAEL, J. AND SHARPEY SCHAFER, E. P.—*Clin. Sci.*, **6**: 187, 1947.

KETY, S. S. AND SCHMIDT, C. F.—*J. Clin. Invest.*, **27**: 484, 1948.

KOUNTZ, W. V. AND ALEXANDER, H. L.—*Medicine*, **13**: 251, 1934.

LENEGRE, J. AND MAURICE, P.—*Brit. Heart J.*, **10**: 83, 1948.

MCILROY, M. B. AND CHRISTIE, R. V.—*Thorax*, **7**: 291, 1952.

MCMICHAEL, J.—*Schweiz. med. Wchnschr.*, **76**: 851, 1946.

MCMICHAEL, J.—Edinburgh Post-graduate Lectures in Medicine, Vol. V, 1952, Oliver & Boyd, Edinburgh, p. 61.

MOTLEY, H. L., COURNAUD, A., WERKO, L., MIMMELSTEIN, A. AND DRISDALE, D.—*Am. J. Phys.*, **150**: 315, 1947.

MOUNSEY, J. P. D., RITZMANN, L. W., SILVERSTONE, N. J., BRISCOE, W. A. AND MCLIMARE, G. A.—*Brit. Heart J.*, **14**: 153, 1952.

PATTERSON, J. L. JR., HEYMAN, A. AND DUKE, T. W.—*Am. J. Med.*, **12**: 382, 1952.

RASMUSSEN, R. A. AND ADAMS, W E.—*Arch. Int. Med.*, **70**: 371, 1942.

RICHARDS, D. W.—*Am. J. Med.*, **3**: 434, 1947.

RICHARDS, D. W. JR.—*Bull. N. Y. Acad. Med.*, **26**: 384, 1950.

RILEY, R. L., HIMMELSTEIN, A., MOTLEY, H. L., WEINER, H. M. AND COURNAUD, A.—*Am. J. Phys.*, **152**: 372, 1948.

SIMPSON, T.—*Brit. M. J.*, **2**: 369, 1948.

WESTLAKE, E. K. AND KAYE, M.—*Brit. M. J.*, **1**: 303, 1954.

CASE NOTES

FATAL FAT EMBOLISM FOLLOWING CRIMINAL VIOLENCE

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In the literature of trauma, a clear cut place is conceded now to fat embolism complicating injury, especially fractures of the long bones. But reports of fatal cases in which the fat originates from subcutaneous fat resulting from criminal violence to the part are so rare that there is enough justification for publishing the following case.

CASE REPORT

The deceased was sleeping on the pavement in front of his house in Chintadripet, when, between 12 midnight and 1 o'clock during the night of 18-8-53, a few policemen in mufti arrived there and without prior warning conducted a raid in front of his house. The deceased was caught in the melee and was reported to have sustained serious injuries. At 1-20 A.M. the same night, the deceased was examined at the Casualty Department of the Government General Hospital and was admitted for treatment of multiple injuries. The deceased was apparently quite well and was progressing satisfactorily and according to the medical attendants, there was no need for recording his dying declaration. But suddenly his condition became bad on the evening of 23rd, difficulty in breathing set in and the patient expired the same night at 9 o'clock.

Autopsy findings: (1) Well nourished individual, about 23 years old. (2) There were 51 wounds from head to foot, consisting of abrasions, contusions and lacerations. There was evidence of infection in many of them. Several of the contusions were noticed on the back of the trunk, arms, buttocks, thighs and scalp. (3) Heart—10 oz.; all chambers contained clotted and fluid blood. (4) Lungs—left 12 oz., right 1 lb. Both were markedly congested and moist. Petechial haemorrhages were present and one area $2'' \times 1''$ was noticed over the middle of the posterior part of the base of the left lung with appearances of haemorrhagic consolidation. (5) Other organs revealed nothing specially abnormal except a scar in the liver. It is worth mentioning that no haemorrhage was seen in the brain.

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Microscopic examination—Paraffin sections of the lung showed marked congestion and oedema. Frozen sections revealed the presence of a number of fat globules in the alveolar vessels. Sections of other organs, spleen, kidney and liver, showed marked congestion. Marked inflammatory changes following pyogenic infection were seen in the sections from scalp wounds and leg wounds.

Alcohol was detected in stomach contents only.

Death was due to pulmonary fat embolism.

Comment

Opinions as to the incidence of fat embolism vary considerably. Out of 164 cases, of varying types of injuries, evidence of severe degrees of fat embolism was found in 11, three proving fatal (Vance, 1931). Fat embolism was considered the major lethal factor in 25 per cent of a series of 115 accidents (Robb-Smith, 1941). Eight cases of clinical fat embolism with six deaths have been described (Wilson and Salisbury, 1944) in a study of 1,000 consecutive battle casualties admitted to the surgical wards of a military hospital.

The condition may be caused in different ways (1) by an injury to adipose tissue which forces liquid fat into the blood vessels. This may be in fractures of long bones or in orthopaedic operations, or in injury to subcutaneous and visceral fat following contusions or burns, (2) by unknowingly injecting oil into the circulation when oil is introduced into the uterine cavity as an abortifacient and (3) by the result of a natural disease process without trauma as seen very rarely in sickle cell anaemia. Mobilized fat, patent disrupted veins and local pressure are essential for the entry of fat into the circulations in traumatic cases. These factors are particularly operative in bone trauma. "Fat is set free from the marrow cells. Disruption of veins and venules without opportunity for collapse of the walls is the rule. As haemorrhage, oedema and exudation develop about the fracture, the local pressure is greater than the venous pressure. Consequently, it is easy for fat to find its way into the disrupted venous channels" (Warren, 1946). The physical state of the fat in the blood is much more important than the total amount. The normal blood fat varies from 6 to 7 per cent. In diabetic coma, where the total blood lipid may reach even 20 per cent, no case of fat embolism has been noticed. The fat present in embolism, is evidently still fluid and when autopsy is done when the body is still warm, the fat is found in a fluid state. The estimate of the minimal amount of fat entering the circulation of man to produce death, ranges from 12 g. to 120 c.c.

There is difference of opinion about the clinical course. From 8 to 20 hours after the accident, the rates of respiration and pulse begin to increase and death from pulmonary embolism may occur in severe dyspnoea about 30 hours after the trauma (Gonzales *et al*, 1954).

According to Wilson (1946), the onset of pulmonary embolism usually occurs during the second or third day after the trauma, but may arise within a few hours and usually not later than the fifth day. Signs of cerebral fat embolism and other systemic embolism set in. Coma may be an early feature followed by increased reflexes and spasticity going on to paralysis of various groups of muscles. There is often irregularity of the pupils and interference with the light reflexes. "About 16 to 48 hours after the injury the patient exhibits a large number of small petechial haemorrhages, grouped in small clusters in the skin of the shoulders, neck, upper chest and in the conjunctivae. At the same time he lapses into a coma which deepens steadily until his death occurs from one to five days later" (Gonzales *et al*, 1954). The clinical picture is thus rather indefinite. Certain tests like examination of blood, sputum, urine and lung puncture have been suggested; but none of these is absolutely conclusive. Hence the clinical diagnosis is rather indefinite and often made by a process of exclusion. "If however, a patient who has had no symptoms other than those due to his injury, suddenly, during the first few days of his illness, develops symptoms such as increased temperature, respiration and pulse rate, is covered by sweat, seems restless and irrational, then the diagnosis of fat embolism is almost certain" (Wilson, 1946).

The gross and microscopic evidence present a wide range of variations. The lungs are usually oedematous, hyperaemic and focally haemorrhagic. In cerebral fat embolism, the brain may be filled with small petechial haemorrhages confined strictly to the white matter. On microscopic examination, fat droplets are detected in the small vessels and capillaries of most tissues. They predominate in the lungs and in the glomerular tufts of the kidney. Frozen sections of fresh autopsy material stained with Sudan III or other special fat stains demonstrate the condition microscopically. In paraffin sections, the presence of vacuoles in the alveolar capillaries or glomerular capillaries is highly diagnostic. The quantity of fat in the pulmonary capillaries ought to be roughly assessed before giving a definite opinion that fat embolism was the cause of death.

While cases of fat embolism associated with fracture of long bones are well known and often reported, cases of fatal fat embolism following injury to subcutaneous fat are extremely rare. One such case was reported by Dutra (1952). It was the case of a woman beaten to death with a chair leg by her husband. Though over 200 distinct marks of injuries were noted on her person there was no single fatal wound. There was no anatomic evidence of protracted shock after autopsy, but fat stains of the lung demonstrated widespread pulmonary fat emboli from the subcutaneous fat that had been liberated by severe beating. No similar case has been reported so far in India to my knowledge. The case briefly described above is one such rare unpredictable and unfortunate instance of death resulting from criminal violence to subcutaneous fat.

References

DUTRA, P.—*Proc. Am. Acad. Forensic Sc.*, **1** : 328, 1952.

GONZALES, T. A., VANCE, M., HELPERN, M. AND UMBERGER, C. J.— Legal Medicine, Pathology and Toxicology, II Edition, Appleton Century Crofts, New York, 1954.

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References

DUTRA, F.—*Proc. Am. Acad. Forensic Sc.*, **1** : 328, 1952.

GONZALES, T. A., VANCE, M., HELPERN, M. AND UMBERGER, C. J.— Legal Medicine, Pathology and Toxicology, II Edition, Appleton Century Crofts, New York, 1954.

ROBB-SMITH, A. M. T.—*Lancet*, **1** : 135, 1941.
VANCE, B. M.—*Arch. Surgery*, **23** : 426, 1931.
WARREN, S.—*Am. J. Path.*, **22** : 70, 1946.
WILSON, J. V.—The Pathology of Traumatic injury, 1946, E. S. Livingstone, Edinburgh.
WILSON, J. V. AND SALISBURY, C. V.—*Brit. J. Surg.*, **32** : 384, 1944.

TWO CASES OF ERYTHROBLASTIC BABIES OF WHOM ONE WAS SAVED BY EXCHANGE TRANSFUSION†

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Rh sensitization of mothers in the Indian population is not uncommon. Five cases of iso-immunization of mothers have already been reported from Calcutta (Greval, Raychowdhury and Banerji, 1946 and 1947; Greval, Roychowdhury and Mukherji, 1948), six cases from Bombay (Sanghvi and Khanolkar, 1947) and two cases from Delhi (Ranganathan, 1947a and 1947b).

In all but two of these cases (Greval *et al, loc. cit.*) the iso-immunization of the mother was associated with erythroblastosis foetalis.

It was, however, only in one case (Ranganathan, 1947b) that the mother's serum was actually demonstrated to agglutinate the baby's cell.

Two cases of erythroblastosis foetalis are reported here where the mother's serum was shown to agglutinate baby's cell.

The sensitization of baby's cell by the Rh agglutinin in mother's serum was also demonstrated in both these cases by the direct anti-human globulin test which per haps is the most direct and positive proof of the affection of the baby. The new-born baby was severely jaundiced in the first case and a partial exchange transfusion from a suitable Rh negative donor was made within 10 hours of delivery and was repeated on the third day. The baby made a good recovery and was in good health up to 10 months when it was reported to have died of an abscess in the neck.

In the case of the 2nd baby, though sensitization of the cells was demonstrated by the direct anti-human globulin test, the reaction was weak and the agglutinin titre of the mother's serum was low. So an immediate transfusion was not attempted and the baby died on the 10th day.

The details of the cases and the results of the serological examination follow.

CASE REPORTS

Case 1—Mrs. S. R., Bengali, H. F., aged 28 years, general condition weak, no history of blood transfusion, urine clear, anaemia slight, oedema nil, weight 108 lbs., height 5 ft., admitted at 33rd week in Eden Hospital, Calcutta, on 14-3-51.

Obstetric history : First issue, 10 years back, at 9th month, died after 8 days. Second issue, 5 years back, at 9th month, died after 2 days. Third issue, 4 years back, at 10th month, died after ½ hour. Fourth issue, 2½ years back, at 10th month, died of jaundice after 3 days. Parents could not give any history of jaundice in the first 3 children. All easy labours except first.

Husband—WR negative ; VDRL negative ; Kahn negative ; Group B ; Rh genotype† R_1r, (C+D+E−c++).

Mother : Serological test—at 28th week—Tests done while attending the out-patient department show : WR negative ; Blood group B ; Rh genotype rr or cde/cde, (C−D−E−c++^{v}). Albumin or blocking agglutinin probably anti Rh_1 (anti C and anti D) was present ; agglutination, +, with husband's R_1r cell.

Serological test at 31st week—It shows that mother's serum did not contain any saline agglutinin anti Rh_1 even when treated with homozygous Rh_1 cell. The serum, however, contained albumin agglutinin against both R_1R_1 and R_0r cells of which R_1R_1 cell gave a stronger agglutination as shown by 2 plus reaction in 1 :64 dilution of serum as against one plus reaction by R_0r cell in the same dilution of the serum.

Serological test at 33rd week—It shows that the mother's serum contained saline agglutinin anti Rh_1, titre 1 :2, detected with $O.R_1R_1$, homozygous Rh_1, cell. The mother's serum also contained an albumin or blocking agglutinin anti Rh_1, titre 1 :2048, with an $O.R_1R_1$, homozygous Rh_1, cell ; 1 :512, with husband's $B.R_1r$, heterozygous Rh_1, cell ; and beyond 1 :128 with $B.R_0r$, heterozygous Rh_0, cell.

The rising titre of albumin agglutinin was shown by the++^{v} agglutination of the undiluted serum of the mother with her husband's cell as compared with + agglutination at the 28th week. The results of the** Coombs test (indirect antihuman globulin test) are detailed in Table 1. The albumin agglutinin contained in the mother's serum sensitized the cells containing the genes R_1 and R_0 and not the gene r. The degree of sensitization was also quantitative and was in the order of homozygous Rh_1, heterozygous Rh, and heterozygous Rh_0 cells. The gradation, if not accidental, is a sufficient new finding.

† A resume of this paper was read in the Medical and Veterinary Section of the 41st session of the Indian Science Congress Association, Hyderabad, 1954.

† Genotype in this paper means "probable genotype" only.

** Reading of slide agglutination (Greval and Roychowdhury, 1946).

TABLE I—SHOWING THE ANTIBODY IN THE SERUM OF MRS. S. R. AT 33RD WEEK AS DETERMINED BY INDIRECT ANTI-HUMAN GLOBULIN TEST (COOMBS TEST)

Cells used in the test	Anti-human globulin serum		Sensitized cell (Control of the test)
	Sensitized cell (Test proper)	Unsensitized cell (Control of cells)	
O.R_1R_1 (S.R.M.) ...	+ +	–	–
B.R_1r (Husband) ...	+(+)	–	–
B.R_0r (J.C.S.) ...	+	–	–
O. rr (M.D.) ...	– (serves as negative control of serum)	–	–

The agglutination with R_1r cell was of a higher order than with R_0r cell proving that the agglutination in question was really anti Rh_1 and not anti Rho. This confirmed the conclusion drawn on the result of titration.

Baby: at the 37th week (14-5-51) a female child was delivered by caesarean section, placenta normal, baby anaemic, slightly yellow tinged.

Cord blood: Colour of serum—highly jaundiced. Bilirubin content—3·6 mg. per 100 ml. Haemoglobin—8 g. per 100 ml. RBC—2,750,000 per c.mm. WBC—7,000 per c.mm. Immature cells—60 per 100 leucocytes. Group—B. Direct anti-human globulin test—+ +. Rh genotype—R_1r [C(+)D(+)E–c(+)]. RBC (suspended in saline) tested with mother's serum, both being of the same group, on a slide—+.

An instrument was improvised for exchange transfusion in collaboration with the blood transfusion officer, provincial blood bank.

Transfusion: The same day the baby was born an exchange transfusion of 200 cc. of Orr blood (donor M.D.) by umbilical vein was given. Towards the end when blood could not be satisfactorily withdrawn through the umbilical vein it was let out by cutting one radial artery. On the 3rd day a second transfusion of 175 cc. of Orr blood (donor S.B.) was given and 45 cc. of the baby's blood let out by cutting a brachial artery.

Progress of the baby: To judge the progress of the baby by determining the fate of the original and transfused cells direct anti-human globulin test and differential isohaemagglutination were carried out at different stages. The method followed was of slide technique. The findings have been tabulated (Table 2).

Follow up of the baby: Baby was keeping good health and growing normally up to the 10th month when she died of an abscess in the neck.

Case 2—Mrs. B. R. B., Bengali, H. F., aged 23 years, admitted on 23-10-51 in Eden Hospital, 5th gravida, carrying 32 weeks I.OA, head engaged, FHS+, general condition average, Hb. 70 per cent, jaundice icteric tinge, oedema nil, liver 2 fingers soft, urine scanty, albumin+, BP 120/80, no history of previous blood transfusion.

Obstetric history: 1st issue, 9 years ago, full term, died after 3 or 4 days. 2nd issue, 6 years ago, full term, living. 3rd issue 4½ years ago, died of jaundice after 10-12 days. 4th issue, 3 years ago, miscarriage at the 5th month.

Husband: WR negative, Group B, Rh genotype R_1R_1, (C+ +D+ +E–c–).

Daughter: Miss K.R.B., only surviving child, W.R. not done, Group A_1B, Rh genotype R_1r, (C+ +D+ +E–c+ +).

Mother—Serological test at 32nd week showed: WR negative, Group A_1B, Rh genotype rr or cde/cde, (C–D–E–c+ +).

The results of antibody determination by titration are detailed in Table 3. Albumin or blocking agglutinin anti Rh_1, titre 1:4 with $A_1BR_1R_1$ cell and also husband's cell, BR_1R_1. No saline antibody could be detected. The titre *in vivo* fell to 1:2 three days after delivery.

As the titre was insignificant in comparison with the previous case it was decided not to give an exchange transfusion straightway but to watch the condition of the baby by checking direct anti-human globulin test and haemoglobin estimation.

Baby: Baby was born on 8-11-51 unexpectedly prematurely at 34th week, placenta and membrane 1 lb., cord 18″, baby female, asphyxiated, wt. 6 lb.—8 oz., length 19″.

Cord blood was not collected.

Blood taken by heel stab showed: Colour of serum—only slightly jaundiced. Abnormal cells—52 normoblast C per 100 leucocytes. Group—A_1. Rh. genotype—R_1r, (C+D+E–c+). Direct anti-human globulin test—Weakly positive, (+). RBC suspended in serum and mixed with mother's serum (being compatible) on a slide—+(+).

Progress of the baby: Determined by repeated direct anti-human globulin test and haemoglobin determination to decide the advisability of transfusion. The figures have been tabulated (Table 4).

It will be evident from Table 4 that advisability of transfusion can be judged more precisely by direct anti-human globulin test and haemoglobin estimation than clinically. Differential agglutination might have supplied similar information.

TABLE 2—SHOWING FATE OF ORIGINAL AND TRANSFUSED CELLS

Cell of baby at different stages	Direct antihuman globulin test (read with unaided eye)	Isohaemagglutination with agglutinin 'b' (read under microscope after 1 hr., notations are same as those of Rh tube technique)	Remarks
Immediately after birth, cord blood.	++, Practically no free cells. (Begins in 15 sec., starts centrifugal in 60 sec.)	W, 'W' clump all over. (Begins in 120 sec.)	All cells blocked by Rh antibody. Group B antigen probably not fully developed.
3rd day after 1st transfusion, or 3rd day of birth.	+(+), Some free cells. (Begins in 30 sec., starts centrifugal in 60 sec.)	(+), Fewer but slightly larger clumps. (Begins in 60 sec.)	Some of the cells are replaced by transfused cells.
3rd day after 2nd transfusion, or 5th day of birth.	(+), Mostly free cells.	+, Fewer but still bigger clumps.	More cells are replaced. Number of original cells being less agglutinin 'b' can act better.
9th day of birth ...	– (Negative).	W, Except few small clumps all cells are free.	Practically all cells are transfused cells.
13th day of birth ...	– (Negative).	25-30 clumps of small and moderate sizes.	Do.

TABLE 3—SHOWING ANTIBODY IN THE SERUM OF MRS. B. R. B. AT 32ND WEEK AS DETERMINED BY TITRATION
(The first dilution showing a 'w' reaction is taken as indicative of titre)

Mother's serum (for albumin antibody diluted with serum from an AB.R′R′ person)	Saline suspension		Own serum suspension			
	Husband $B.R_1R_1$	S.R.D.S. $A_1B.R_1R_1$	Husband $B.R_1R_1$	Same after 2-3 min.	S.R.D.S. $A_1B.R_1R_1$	Same after 2-3 min.
Undiluted	–	–	+	–	(+)	(+)
1/2	–	–	+	(+)	(+)	(+)
1/4	–	–	(+)	w	(+)	w
1/8		–	(+)	w	(+)	w
1/16		–	w	?–	w	–
1/32		–	w	?–	w	–
1/64		–	?–	–	–	
1/128		–	–	–	–	
Control of diluent serum (from an AB.R′R′ person)						
Undiluted	–	–	–	–	–	–

DISCUSSION

The difference in the titre of the albumin agglutinin in the blood of the mother in Case 1 with OR_1R_1 cells and BR_1R_1 cells were not due to anti O in the mother's serum as the control of the test with O. rr cell did not show any agglutination. It might be due to difference in the geno types of the cells, the double dose of antigen of a homozygous cell giving a better agglutination than the single dose of a heterozygous cell. Non-agglutination of the husband's cell by the saline agglutinin present in mother's serum might be due to the same reason, i.e., the agglutinin was too weak to agglutinate the single dose, i.e., weak antigen of a heterozygous cell.

Uniformity of the titre in Case 2 with both the cells $A_1BR_1R_1$ and BR_1R_1 (husband's) might be due to the fact that both the cells were homozygous.

In case of the second baby though the baby did not show any bad sypmtom the direct anti-human globulin test could detect the increase of the harmful effect of the anti-Rh body on the baby's cells up to the 5th day.

TABLE 4—SHOWING PROGRESS OF THE BABY AS DETERMINED BY ANTI-HUMAN GLOBULIN TEST AND HB. ESTIMATION

Cells of the baby at different stages	Direct anti-human globulin test	Haemoglobin	Condition of the baby clinically
Immediately after birth ...	Weakly positive, (+)	—	—
2nd day	Same, (+)	100% (Sahli) = 14 gramme.	Baby was taking food quite satisfactorily but later became bad.
5th day	Stronger agglutination than before, +	75% (Sahli)	Transfusion advised but not given.
8th day	Negative, − (N.B. Also no antibody could be detected in baby's serum though it could be detected earlier)	85% (Sahli)	After this the baby was progressing steadily, but suddenly turned bad and died on the 10th day.

The harmful effect was also shown by the fall in the value of haemoglobin. So these may be taken as better guides than the clinical symptoms. The baby of course died even after the direct anti-human globulin test became negative probably due to inability to recover from the harmful effect already produced by the antibody.

Direct anti-human globulin test was positive on the 5th day and became negative on the 8th or 9th day. In the second baby direct anti-human globulin test gave a weak reaction initially to become stronger later.

Indirect anti-human globulin test with proper control done for antibody determination may give valuable data.

SUMMARY

Two cases of erythroblastosis foetalis are reported here where the mother's serum was shown to agglutinate the baby's red cells.

The sensitization of baby's cells by the Rh agglutinin in the mother's serum was also demonstrated in both these cases by the direct anti-human globulin test. The titre of agglutinin albumin anti-Rh_1 in the mother's serum was 1 :2048, at the 33rd week, with a specimen of OR_1R_1 cells. The new-born baby was severely jaundiced in the first case and a partial exchange transfusion from a suitable Rh-negative donor was made within 10 hours of delivery and was repeated on the third day. The baby made a good recovery and was in good health up to 10 months when she was reported to have died of an abscess in the neck.

In the case of the 2nd baby the direct anti-human globulin test on baby's cells gave a weak reaction and the agglutinin titre of the mother's serum was low, 1 :4. So an immediate transfusion was not attempted and the baby died on the 10th day.

In the first case the antibody gave a higher titre with R_1R_1 (homozygous) cells than with husband's R_1r (heterozygous) cells.

The progress of the babies was determined by the direct anti-human globulin test, differential agglutination and haemoglobin estimation.

This was the first authenticated exchange transfusion carried out in India.

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REFERENCES

COOMBS, R. R. A., MOURANT, A. E. AND RACE, R. R.—*Brit. J. Exper. Path.*, **26** : 255, 1945.

Idem—Lancet, **1** : 264, 1946.

GREVAL, S. D. S. AND ROYCHOWDHURY, A. B.—*Indian M. Gaz.*, **81** : 354, 1946.

GREVAL, S. D. S., ROYCHOWDHURY, A. B. AND BANERJI, B.—*Ibid*, **81** : 243, 1946.

Idem—Ibid, **82** : 76, 1947.

GREVAL, S. D. S., ROYCHOWDHURY, A. B. AND MUKHERJI, N. G.—*Ibid*, **83** : 370, 1948.

RANGANATHAN, K. S.—*Ibid*, **82** : 269, 1947a.

Idem—Ibid, **82** : 404, 1947b.

SANGHVI, L. D. AND KHANOLKAR, V. R.—*Indian J. M. Sc.*, **1** : 45, 1947.

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SCHISTOSOMIASIS IN INDIA

Schistosomiasis, though not usually an acute fulminating ailment marked by dramatic sequence of events as in an acute bacterial or viral infection, is notorious for its cumulative mischief, that goes to exact a devastating economic toll in areas, where it is endemic. According to Stoll's[1] assessment, the world harbours about 114,400,000 individuals with schistosomiasis. This dreadful menace to the public health has been engaging increasing attention of the workers in different parts of the world in recent years. Three species of blood-flukes, e.g., *S. haematobium, S. mansoni,* and *S. japonicum* have generally been held responsible for human schistosomiasis, the site of affection being mainly vesical with the first one, and chiefly intestinal with the last two, while atypical or protean lesions and various combinations are met with less frequently. The human beings can only be infected as a result of contact with water containing the infective cercariae of the blood-flukes, liberated by their molluscan intermediate hosts. The distribution of schistosomiasis, actual and potential, therefore, depends on the availability of the appropriate snail-vector, the reservoir host, chiefly man, and the factors, occupational, sanitary or otherwise, facilitating the contact between man and the offending agent. The epidemiology of the disease has been further complicated with the advent of the modern transport system, the new agricultural techniques, and the development of new water resources for the purpose of irrigation, power projects and human consumption.

The probable existence of human schistosomiasis in India has been a problem haunting the parasitologists since the beginning of the present century. During the period between 1903 and 1948 as many as ten authentic cases of urinary schistosomiasis have been recorded by Powell[2], Sewell[3], Christophers and Stephens[4], Wardrop[5], Hooton[6], Harkness[7], de Mello[8], Andreasen and Suri[9], de Sa and Monteiro[10], from different parts of India, e.g., Bombay, Madras, Goa, Poona, the Punjab and the area now called Saurashtra. Some of these cases have been believed to be definitely autochthonous, and in all the instances *S. haematobium* has been incriminated as the offending parasite. The discovery of *S. incognitum* by Chandler[11] in Bengal, could not receive the recognition as an instance of human infection, because of the doubtful method of stool collection and the subsequent finding of the same species of schistosome in pigs. Investigators have been busy during this period to determine if India runs the risk of being an endemic zone of schistosomiasis. The fact that easy and unabated import of infections by military as well as civilian population from the known endemic areas did not result in any serious outbreak of the disease in India, went a long way to undermine the possible and potential endemicity of India for schistosomiasis. The lack of spread of the disease was attributed by Sewell[12] and others to the probable absence of the optimum snail-vector necessary for the transmission of the infections. This derived quite an able support from the extensive experiments of Kemp and Gravely[13] and Mukherji *et al*[14] to infect various species of molluscs with the miracidia of *S. haematobium*, yielding negative results. But de Mello[15] from attempts to infect some fresh water molluscs observed that miracidial attraction was very active and often followed by penetration. He also found a natural case of infection in *Melanoides* with furcocercous cercariae, indistinguishable from those of *S. haematobium*. Bhalerao[16], however, was of opinion that the clinical, epidemiological and experimental data, obtained so far, did not warrant the conclusion that urinary bilharziasis might become endemic in India. But the impression gaining ground so long began to dwindle away fast with the discovery of an endemic focus in the

[1] STOLL, N. R.—*J. Parasitol.*, **33** : 1, 1947.
[2] POWELL, A.—*Brit. M. J.*, **1** : 490, 1903.
[3] SEWELL, E. P.—*J. Roy. Army. Med. Corps.*, **2** : 346, 1904.
[4] CHRISTOPHERS, E. AND STEPHENS, J.—Quoted by Bhalerao (16).
[5] WARDROP, D.—*J. Roy. Army Med. Corps.*, **7** : 282, 1906.
[6] HOOTON, A.—*Indian M. Gaz.*, **49** : 188, 1914.
[7] HARKNESS, A. H.—*Brit. M. J.*, **1** : 475, 1922.
[8] DE MELLO, I. F.—Quoted by Bhalerao (16).
[9] ANDREASEN, A. T. AND SURI, H. L.—*Indian M. Gaz.*, **80** : 93, 1945.
[10] DE SA, A. E. AND MONTEIRO, L.—*Indian J. M. Sc.*, **3** : 376, 1949.
[11] CHANDLER, A. C.—*Indian J. M. Research*, **15** : 179, 1926.
[12] SEWELL, R. B. S.—*Indian M. Gaz.*, **54** : 252, 1919.
[13] KEMP, S. AND GRAVELY, F. H.—*Indian J. M. Research*, **7** : 251, 1919.
[14] MUKHERJI, A. K., BHADURI, N. V. AND NARAIN, S.—*Ibid*, **43** : 311, 1946.
[15] DE MELLO, I. F.—*Proc. Indian Acad. Sc.*, **3** : 107, 1936.
[16] BHALERAO, G. D.—*Proc. 35th Indian Sci., Cong.*, Part II, p. 57, 1948.

district of Ratnagiri of Bombay State by Gadgil and Shah[17], who recorded the infection in 250 individuals in a population of about 1,200. But the search for the appropriate intermediate host is yet to be called a complete success, though the authors found cercariae resembling those of *S. haematobium* in 8 of 300 snails dissected. The same authors[18, 19] have undertaken the fascinating study of snail population and experimental transmission of schistosomiasis in recent years, and have reported that the snail, *Ferrissia tenuis* can be infected with the miracidia of *S. haematobium*. But before this snail is accepted as the optimum vector, it is essential that the infection be transmitted with ease to some experimental animals and the identity of the adult parasite developing therein be definitely established to be that of *S. haematobium*. It is, however, in the fitness of the things to mention here, as pointed out by Verma[20], that the possibility of accidental human infections with various animal schistosomes, not known to have any marked degree of host specificity, and with terminal spine ova bearing close resemblance with that of *S. haematobium*, requires to be dismissed, before the identity of the adult schistosomes infesting man can be precisely known, particularly, when more than one species of animal schistosomes, e.g., *S. bomfordi*[21], S. *spindalis* and *S. indicum*[22], etc. have already been reported to be occurring in India.

The current issue of the journal contains a report by Dhanda[23] of four cases where examination of stool revealed the presence of schistosome ova with terminal spine, resembling those of *S. haematobium*. No such ova could be found in the urine and no other evidence of urinary tract involvement was available. It should be remembered, however, that the examination of the terminal 2 or 3 c.c. of urine and urine after severe exercise offers the greatest chance of detecting schistosome ova. Though the study of Gelfand and Ross[24] from 110 autopsies of bilharzial subjects revealed the frequent association of intestinal tract involvement in infection with *S. haematobium*, the affection of intestinal tract only by *S. haematobium* is to be regarded conspicuously rare. Fairley[25] while dealing with susceptibility of man to cattle schistosomes observed that it is quite difficult to differentiate on morphological grounds ova of certain species of cattle schistosomes from *S. haematobium* in the excreta. This is also readily referable to the extreme difficulty, pointed out by Amberson and Schwarz[26] in the precise identification of the species of schistosome from the ova with terminal spine, where one has to deal with a graded series of forms with overlapping character. They observed, "This variation may produce extreme individual types within a population not distinguishable from those typical of another form". In this group they included *S. haematobium, S. capense, S. intercalatum, S. mattheei* and *S. bovis*. The last two parasites, of course, require further experimentation to be definitely established as potential human parasites. But, *S. intercalatum*, has so far been reported to be entirely restricted to man causing intestinal symptoms only.

Therefore, the possibility that the infection in the present series might be due to other species of schistosome, viz., *S. intercalatum*, or any of the animal schistosomes with terminal spine ova, should not be completely ignored.

For a correct appraisal of the schistosomiasis problem in India an extensive investigation should be undertaken, which must include, in addition to the meticulous quest of possible endemic foci, an accurate and sound assessment of the taxonomy of the species of flukes involved and that of the snail likely to serve as intermediate hosts. To be of scientific value, the results of such a reconnaissance should have corroboration from experimental transmission. The knowledge thus acquired will allow a clear understanding of the epidemiology and the clinical picture of the disease and help in the formulation of effective therapeutic and control measures. The problem of schistosomiasis also calls for an intensive study of the biology and physiology of the parasite and the intermediate host and the possible factors influencing their mutual relationship. The valuable information that will accrue from these studies will contribute immensely towards the eradication of this formidable enemy of human race.

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[17] GADGIL, R. K. AND SHAH, S. N.—*Indian J. M. Sc.*, **6**: 760, 1952.

[18] *Idem—Ibid*, **9**: 586, 1955.

[19] *Idem—Ibid*, **9**: 592, 1955.

[20] VERMA, A. K.—*J. Indian M. A.*, **25**: 173, 1955.

[21] BOMFORD, G.—*Scient. Mem. M. Officers, India*, Part II, p. 53, 1886.

[22] MONTGOMERY, R. E.—*J. Trop. Vet. Sc.*, **1**: 15 & 138, 1906.

[23] DHANDA, LEILA—*J. Indian M. A.*, **26**: 407, 1956.

[24] GELFAND, M. AND ROSS, W. F.—*Tr. Roy. Soc. Trop. Med. & Hyg.*, **47**: 215, 1953.

[25] FAIRLEY, N. H.—*Tr. Roy. Soc. Trop. Med. & Hyg.*, **45**: 279, 1951.

[26] AMBERSON, J. M. AND SCHWARZ, R.—*Ibid*, **47**: 451, 1953.

CURRENT MEDICAL LITERATURE

The Choice of Sedatives in Old Age

RUDD (*Practitioner*, **176**: 218, 1956) writes:

Broadly speaking, sedation is required for the quietening of a restless, lucid patient, or for the control of a confused one. In old age, the borderline between lucidity and confusion is a narrow one, easily passed in either direction. Restlessness is the elderly, with its counterpart, senile confusion, can occur in any of the following states: 1. Nervous agitation. 2. Bodily discomfort from inactivity, pain, thirst, overheating, rectal or vesical distension. 3. Metabolic upsets—fever, toxaemia, electrolyte imbalance, avitaminosis. 4. Functional or organic cerebral upset—congestion or anaemia, cerebrovascular accidents or inflammation, cerebral tumours. 5. Drug intoxication, especially after bromides, barbiturates, morphia derivatives, hyoscine, and sulphonamides.

In approaching the problem, these causes require consideration, and a brief physical examination should be made to identify the probable operative factor. Initial treatment should be directed to restoring bodily comfort, to emptying a loaded bladder or bowel, tepid sponging and analgesics. Passive venous congestion of the brain from cardiac failure should be treated with a mercurial diuretic, which often relieves restlessness much more effectively and speedily than any sedative.

If no cause for the restlessness is discoverable, or if general measures fail to control it, recourse to drugs is required.

Choice of drugs—Milder cases of restlessness can be tried out first with alcohol, tablets of aspirin and Dover's powder of 10 grains each, *methylpentynol* ('oblivon')—two to five 250 mg. capsules or 120 to 130 minims of the elixir, or *'doriden'*—two 0·25 g. tablets at night.

None of these drugs is likely to influence the severer cases of restlessness, for which early recourse to *paraldehyde* is indicated. Paraldehyde is a safe and reliable sedative, best administered by intramuscular injection of 5 to 8 ml. Further injections of the same, or slightly lower, dosage can be repeated every four to six hours as required. The solution is self-sterilizing, but hydrolyses in the ward stock bottle to glacial acetic acid. It should therefore be used from 5-ml. or 10-ml. ampoules.

Morphine and hyoscine—When paraldehyde proves ineffective, as at times it does, the much less safe hyoscine, alone or combined with morphine, may be tried. Such obstinate cases, however, are of bad prognosis, and generally indicate grave intracranial disease. Both drugs are unreliable unless given in full dosage, when the lethal dosage is not far off.

This list of sedatives is by no means exhaustive. Chloral hydrate, chloralamide and carbromal have their advocates, but have won little recognition from the most practical workers.

Chlorpromazine—In many cases it is certainly effective in lessening anxiety and nervous tension and in enhancing the effect of other analgesics and sedatives. Chlorpromazine suppositories (100 mg.) have a place in managing the acute case, and one inserted rectally is often most effective. It should be remembered that liver disease, past or present, is a contraindication to chlorpromazine therapy.

Amoebic Ulceration of the Buttock

SAUNDERS (*South African M. J.*, **29**: 741, 1955, Ref. *Trop. Dis. Bull.*, **53**: 55, 1956) reports a destitute 29-year-old African woman who entered hospital with a large ulcer of the left buttock of 2 or 3 months' duration. Conventional treatment, including the use of penicillin and aureomycin, failed to clear it so she was readmitted to hospital some 2 months later; a history suggestive of amoebic dysentery was then disclosed. No proof of an intestinal *Entamoeba histolytica* was forthcoming on stool examination, but a clinical diagnosis of cutaneous amoebiasis was made; this was confirmed on biopsy. Under appropriate specific treatment with emetine and other drugs the condition rapidly healed.

Treatment of Amoebiasis

ADAMS (*Brit. M.J.*, **1**: 288, 1956) at a meeting of the Royal Society of Tropical Medicine & Hygiene on the discussion on the treatment of amoebiasis, speaking of intestinal amoebiasis only, summed up his vast experience—which includes long-continued observations of pensioners—in a forthright statement that for the acute attack nothing had so far equalled emetine, that to eradicate the infection a three-weeks intensive course of emetine bismuth iodide was time well spent, that of the arsenicals acetarsol was probably the best, that retention enemata of chiniofon were sometimes useful, and that the antibiotics had not justified themselves for routine treatment but should be reserved for exceptional cases and then used with care.

Professor A. W. Woodruff and his colleagues Dr. S. Bell and Dr. F. D. Schofield, of the Hospital for Tropical Diseases, London, gave complete details of the treatment and observations of several hundred patients treated during several years on a careful plan to evaluate the various drugs used. Like Dr. Adams, Professor Woodruff was unequivocal that emetine bismuth iodide was at present the drug of choice in chronic intestinal amoebiasis (with emetine itself for the acute attack), and he quoted a known relapse rate of 3 per cent in his series—possibly a rather low estimate in view of the difficulties of prolonged observation, but nevertheless comparing favourably with that with other courses of treatment. This team of workers had also tested chloramphenicol (poor), chlortetracycline (poor), oxytetracycline (unsatisfactory), fumagillin (disappointing), camoform (unsatisfactory) glaucarubin (not so good as emetine bismuth iodide, but useful and cheap), and a new drug entamide (fairly useful and cheap). Some of these drugs had considerable value, but they did not stand comparison with emetine bismuth iodide; nevertheless, lack of toxicity and cheapness were factors. Prolonged use of the antibiotics might be risky.

" Precisely, Mr. Baxter . . .

. . . that puts the matter in a nutshell. One *can* say that I.Z.S. is the insulin of the moment . . . particularly for a new diabetic. The thing to remember, gentlemen, is that I.Z.S. seems to have it both ways, so to speak. It gives you a rapid initial response, yet the one injection will usually control the blood sugar level adequately for up to 24 hours. This, as you will find, is a great advantage to your patient. There are, of course, a few patients who benefit more from a modification of the standard I.Z.S. In such cases, you can vary the proportions of the different forms of I.Z.S. in the injection. If you consider that quicker initial action is desirable, the patient can use more I.Z.S. amorphous in his syringe, or alternatively, add more I.Z.S. crystalline to prolong the effect of the injection. Any other questions ? "

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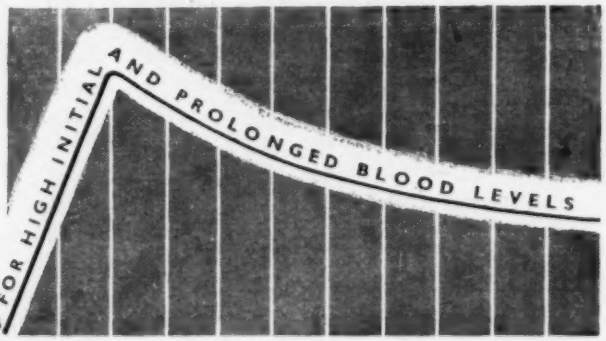

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Treatment of Amoebiasis with Erythromycin

VILLAREJOS (*Am. J. Trop. Med. & Hyg.*, **4** : 699, 1955, Ref. Trop. Dis Bull., 53 : 557, 1956) writes :

Erythromycin is rapidly absorbed from the intestine; but as it is largely destroyed by gastric juice it must be given in an acid-resisting coating. It rapidly diffuses into the tissues from the blood, and thereafter is excreted chiefly in the urine and faeces. Concentrations in the tissues reach higher levels, and persist there for longer, than in the blood; the liver concentrates the drug, which is excreted in the bile in active form; the concentration in the bile is up to 30 times that in the serum. Erythromycin does not cause changes in the normal intestinal flora, and it is virtually non-toxic.

Enteric-coated tablets of erythromycin were given orally to 98 patients in Bolivia with parasitically proven amoebiasis. Twenty-eight of them had acute manifestations of the infection; while 70 suffered chronic, indefinite, or no symptoms resultant on the infection. The initial dosage in all cases was 800 mg.; thereafter 200 or 300 mg. was given every 6 hours or every 8 hours for 5 days (totals of 3·6, 4·6 or 6·5 g.).

The immediate results in the 70 chronic cases was disappearance of parasites from the stools of 9 of the 10 patients given 3·6 g., 22 of the 24 given 4·6 g., and all of the 36 given 6·5 g.; parasitic relapse occurred in 4, 3 and 1 of these respectively, within an average period of observation of about 3 months. Thus the effect in cases of chronic amoebiasis was highly satisfactory when erythromycin was given in the highest dosage used.

The immediate results in the 28 acute cases, all of them passing dysenteric stools, were spectacular in most cases. The symptoms vanished and parasites usually disappeared from the stools within a very few days, and always by the fifth day, whether the dosage given was a total of 4·6 g. or of 6·5 g. Three patients of the 8 on the lower of these dosages relapsed within an average period of two months' observation. Only one patient of the 20 on the higher dosage relapsed; she did so within a week. It seems she had previously had irregular and insufficient erythromycin treatment; this suggests that erythromycin resistance was rapidly developed by her parasites.

Three patients with evidence of amoebic hepatitis were treated with an initial dose of 1 g. and 1·2 g. of erythromycin daily for 7 days, with apparently completely satisfactory results.

Hazards of Chlorpromazine

Minor and often transient side-effects are encountered in most patients receiving chlorpromazine ("largactil"). These include pallor, tachycardia, hypotension, dryness of the mouth, frequency of micturition, and disturbances of bowel action. They are probably due to its action on the autonomic nervous system and seldom make it necessary to stop the administration of the drug. However, a small minority of patients—about 1 per cent—develop toxic reactions such as pyrexia, skin eruptions, oedema, epileptic fits, hepatitis, and agranulocytosis, and it is among this group that fatalities have been reported. When hepatitis has occurred in patients who have received other potentially hepatotoxic drugs, or who had pre-existing liver disease, it has not yet been possible to implicate chlorpromazine with any certainty, but there is now ample evidence that it may cause damage to a hitherto normal liver. The first manifestation is usually jaundice, but this may be preceded by a few days of pyrexia or a prodromal period similar to that seen in infective hepatitis. The combination of pruritus, hepatomegaly, pale stools, and bilirubinuria suggests that the jaundice is of the obstructive type, and this is supported by tests of liver function. Usually the patient completely recovers within a week or two even though administration of the drug is continued, but in some cases jaundice has persisted for many weeks. When death has resulted from hepatic failure, there has usually been evidence of previous liver disease, and chlorpromazine could not be incriminated as more than a contributory cause of death. But chlorpromazine can be implicated directly as a cause of agranulocytosis, and a few deaths have occurred of patients in whom there was no cause to suspect a previous blood dyscrasia.

The mechanism whereby chlorpromazine causes these toxic reactions is obscure. In a series of cases reported by Issacs and his colleagues (*Brit M. J.*, **2** : 1122, 1955) no correlation existed between the onset of jaundice and the total dosage or the dose being given at that time. This would suggest that chlorpromazine is not a direct liver poison. Moreover, it was found that the greatest incidence of jaundice was three to four weeks after the treatment began and in no case did jaundice appear after the drug was stopped. When chlorpromazine was continued despite toxic reactions, spontaneous recovery usually ensued. These findings suggest that the toxic effects may be due to sensitization to the drug. In a second series, jaundice appeared in some cases within a few days of beginning the drug and in others up to two weeks after discontinuing it. Nevertheless it was not always possible to exclude other causes of jaundice in these patients. As to agranulocytosis, future experience may well show that chlorpromazine is to be included in the group of aromatic organic compounds well known to depress the production of leucocytes.

These serious complications call for special care in administration of this double-edged therapeutic weapon. Certainly it is contraindicated by previous liver disease or when infective hepatitis is prevalent. Nor should it be given to patients with a previous history of blood dyscrasia or to patients already receiving drugs known to depress the formation of leucocytes.—Annotation, *Brit. M. J.*, **1** : 391, 1956.

Fatal Agranulocytosis after Chlorpromazine

THERIAULT (*Canadian M. A. J.*, **74** : 148, 1956) in reporting a fatal case of agranulocytosis associated with chlorpromazine observes that although this complication is rare, it must be borne in mind. Patients on chlorpromazine should have frequent white cell counts.

Foreign Body Endoscopy

BALLON (*Canadian M. A. J.*, **74** : 139, 1956) in giving a review of foreign body endoscopy over a period of 30 years writes :

One hundred foreign bodies were removed from the larynx, trachea, bronchi and oesophagus. The youngest patient was a six-week-old infant with an open safety-pin in the oesophagus, and the oldest was 89 years old with a fish bone in the oesophagus.

Of the foreign bodies in the larynx and tracheo-bronchial tree, 60 per cent were in infants under three years of age. The youngest child was four months old, with a rivet in the larynx.

It was not uncommon to overlook a foreign body because of the symptomless interval following its aspiration, and because of errors in diagnosis. It is of vital importance to differentiate between an "aspirated" foreign body and a "swallowed" foreign body. Obstructive emphysema and atelectasis are two x-ray signs which in a child are practically diagnostic of a foreign body.

The vegetable foreign bodies are the most acutely dangerous to life and function. Nine peanuts were removed from the trachea and bronchi. The oesophagus yielded 25 chicken and meat bones, 10 fish bones, five safety-pins, and nine coins.

Ninety-seven patients were cured, including three who required lobectomy for extensive bronchiectasis, produced by the undiagnosed, prolonged sojourn of the foreign body in the bronchus.

There were three deaths. In these cases the foreign bodies were removed but the patients died from complication. The first patient was a morbund 22-month-old child (temperature 106·2°F), with an overlooked tack in his right bronchus for eight days. Another was a 13-month-old child who aspirated a piece of apple into the trachea and left bronchus. Both died from an extensive bronchopneumonia. The third patient was a woman of 48 who swallowed a ragged pork bone, and in whom unsuccessful attempts were made elsewhere to dislodge the bone on two successive days by blind bouginage and the use of emetics. Before oesophagoscopy, the patient had a chill lasting for 15 minutes, and a temperature of 105°F. Six days after removal of the bone she died suddenly from a perforation of the oesophagus and the arch of the aorta.

There is only one treatment for foreign bodies in the food and air passages—early endoscopic removal. It saves life and function and avoids complications.

Portal Hypertension due to Extrahepatic Obstruction

DAS AND BASU (*Brit. M. J.*, **1** : 325, 1956) report four cases of extrahepatic obstruction of the portal system of veins with repeated severe haematemeses and melaena. Two were due to cavernomatous transformation of the portal vein and two to obstruction of the splenic vein by chronic inflammatory pancreatic masses.

In three cases, splenoportal venography clearly demonstrated the sites of obstruction and their possible nature. Both these findings were subsequently confirmed.

In addition to the routine methods of investigation, the advantages of splenoportal venography in such cases, both in diagnosing the cases of haematemesis and in planning the operation, are commented upon.

CURRENT TOPICS

INTERNATIONAL CONFERENCE ON THE PEACEFUL USES OF ATOMIC ENERGY

The first international conference on the Peaceful Uses of Atomic Energy was held at Palais des Nations, Geneva, August 8-20, 1955. Dr. H. J. Bhabha of India presided.

On the suggestion of President Eisenhower, the conference was organized by the United Nations. There were nearly 1300 participants from 70 countries.

The plenary sessions were devoted to topics of general interest, such as World Energy Needs, Building of Nuclear Energy Enterprise, Health and Safety, Use of Isotopes in Technology and Industry, etc.

There were three separate commitees. Committee A discussed reactors, equipment and techniques, measures of atomic energy and nuclear physics.

Committee B discussed chemistry, metallurgy, technology of nuclear energy, and geological considerations of uranium and thorium.

Committee C examined the problems of biology and medicine, and approximately 250 papers were presented in the medical field.

Although research scientists in various countries have worked independently, the conference brought out similarity of work, analogy of clinical study, and similarity in therapeutic methods. Once again it was demonstrated that scientific and medical research knows neither geographic nor political frontiers.

It was brought out that about 60 substances are used as carriers of radio-activity—particularly, iodine, cobalt, boron, gold, sodium, phosphorus, potassium and carbon.

There is general agreement regarding the discoveries and their meanings, both in the diagnostic and therapeutic fields.

The effects of ionizing radiation on the genetics of plants, animals and men have been demonstrated by many scientists, especially Wallace (USA), Russel (USA), and Carter (Great Britain) in human genetics. If protection against ionizing radiation is not an insoluble problem because of effective security measures, there exists on the other hand the problem of radioactive effects on human genetics which bring on serious risks and involve the responsibility of man towards his own destiny.

SECURITY NORMS FOR WORKERS

Due to the dosage of radiation, control of installations and recommendations being followed up in areas where nuclear energy is being used, there do exist security norms. In his report, Letavet (USSR) described how a physical examination is given to workers undergoing ionizing radiations, twice a year in the USSR in a medical centre known as the "health department", on the staff of which there is a therapist, a neuropathologist, sometimes a dermatologist, an ophthalmologist and a gynecologist. This physical examination includes a blood

test, lung x-rays, and a thorough examination of the nervous system, besides the regular clinical examination. Labourers who are capable of working but who must undergo a short treatment are hospitalized in night sanatoria. The maximum dosage radiation acceptable in the USSR is 0·05 roentgen a day, and in the USA, 0·30 roentgen a week.

THERAPEUTICS

Instruments using radioisotopes advantageously replace, in certain cases, the classical equipment of high tension x-rays. Actually, these instruments are not as bad as the others; for example, it has been estimated that 30 grams of radiocobalt, costing approximately $17,500, produce the same results as a quantity of radium costing $50,000,000. This is because radiocobalt has a relatively short period of activity, about 5 years, and it is often replaced by Cesium 137 which has a period of activity of 37 years.

Methods of applying radioactive isotopes are many and the number is increasing every day. At present, these methods are used for neoplastic illnesses and blood diseases.

The last plenary sessions were devoted to a discussion of international co-operation in the peaceful uses of atomic energy, and to the education and training of personnel in nuclear energy.

During the discussion, specialized agencies of the UN presented the following ideas :

The World Health Organization suggested at the conference a project for an international programme covering these 4 points :

(1) Training of sanitary personnel
(2) Dissemination of medical information
(3) Universal study of the effects on the human body, and
(4) A study of hereditary imperfections.

The International Labour Organization insisted on protection for the workers against ionizing radiations and presented a legislative programme.

Finally, UNESCO presented a proposal for perfecting the efforts of research personnel. The conclusions of this report were as follows :

(1) An effort must be made to introduce into the university programmes for the training of research scientists and engineers in the new fields of physics, chemistry and biology.
(2) Practical uses of discoveries should be made known in study centres associated with business organizations because there are too many varieties of such uses for them to be restricted to general instruction.
(3) Future research scientists must increase their basic knowledge in other extra-professional fields; for example, chemists should study radio-activity, mathematicians nuclear physics, etc.
(4) Certain subjects, especially radiobiology, should be compulsory for all research scientists, engineers and technicians who plan to work in the field of atomic energy.

The first conference was held for the purpose of bringing together scientists and experts from all over the world and exchanging scientific information on the subject of atomic energy and the peaceful uses thereof.

Numerous eminent scientists—specialists in the field of atomic science—came from all countries, met in Geneva, and actively participated in the conference; they were able to gather together information, either by studying reports presented at the conference or by cementing relations with colleagues of other countries. It is not an exaggeration to state that the conference was a complete success. This allows us to hope that in the future the dissemination of information and the exchange of scientific data will be greatly facilitated.

Considering the power placed in human hands by the use of atomic energy, the doctors ask humanity to realize the risk involved in such power, and hope that this energy will be limited strictly to peaceful uses.—Reported by Dr. Jean Maystre and published in the *World Medical Journal*, **3** : 109, 1956.

BE A MEMBER OF THE INDIAN MEDICAL ASSOCIATION

At the 3rd annual conference of the Bombay Provincial Branch of I.M.A., the following appeal dated Bombay the 25th March, 1956, by Dr. C. L. Mehta, a past President of the I.M.A., was circulated to those registered medical practitioners who are not yet members of the Association. This appeal reiterated with necessary additions and alterations, during all Provincial Medical Conferences, will go a long way in furthering the cause of the Association besides enlisting new members.—ED., *J. Indian M. A.*

WHY I SHOULD BE A MEMBER OF THE INDIAN MEDICAL ASSOCIATION

Because :—

It is the only All-India Organisation of the medical profession of the country.

Its membership is open to all Indians qualified in modern medicine.

It has on its membership independent medical practitioners, general and specialists, members of medical and public health services, teachers of medical colleges, laboratory and research workers and members interested in social and industrial medicine.

It works on a democratic constitution and has over 500 branches all over the country.

It gives its attention to all problems of interest to the medical profession and to subjects of medical relief and public health in the country.

Because of its all-India contact it is able to collect first-hand information on all questions and is able to bring to bear on its decisions the viewpoints of all parts of the country.

The Government of India and most of the State Governments invite its opinion on a large number of problems.

It is invited to send its representatives to many committees. On some of the statutory committees it has secured a regular representation.

One of our past presidents has been frequently invited to Health Council of India's meetings as an observer.

It is a member of the World Medical Association and its representative is on its Council of Management. He is invited to the W.H.O.'s meetings as an observer. The I.M.A. representative was invited to the Medical Education Conference sponsored recently by the Government of India.

It has a representation on the Employees State Insurance Corporation, Indian Nursing Council and Pharmaceutical Council of India.

Its Journal has reached a stature that is comparable to any of its kind in the world. It is accepted for exchange.

No medical person either in the teaching service of the Government or local bodies or in independent practice can in these days effectively put forth his views or grievances in his individual capacity. No local medical organisation also can be successful in such efforts. It is only an all-India organisation like the Indian Medical Association that can help them in solving their problems.

It is trying to serve the people through its members by maintaining the tradition of service to the sick, of prevention of disease and promotion of health. It publishes two monthly journals for the benefit of the people at great financial loss—one in English called **"Your Health"**, and another in Hindi named **"आपका स्वास्थ्य"** written in easy language.

It helps young medical graduates to get internships in recognised hospitals in U.S.A. and Canada, at a small cost.

Men like Drs. Deshmukh, Ansari, B. C. Roy, K. S. Ray, Rama Rao, Amesur, P. B. Mukerji, Jivraj Mehta, Col. Amirchand and Dr. S. C. Sen have guided the formation and progress of this Association. Dr. A. C. Ukil, Ex-Principal of the Calcutta Medical College is at its helm this year.

The Government of India and the Governments of the States have put no ban on their service members to join the I.M.A. Even the Defence Department has permitted its Medical Men to be members of the I.M.A.

It is through the co-operation of the I.M.A. that the Employees State Insurance Scheme started going well. It is through the efforts of the Bombay Territorial Branch, I.M.A., and the acceptance of its co-operation by the State, the scheme has been working fairly satisfactorily in Bombay.

As every citizen is proud of his National Government and is eager to contribute his mite to its growth and strength, so should be a medical man for his own National Organisation. It is for this that I feel proud of being a member of my profession's National Organisation, and I expect every member should feel that thrill by joining the Indian Medical Association.

CHAMANLAL MEHTA.

NOTES AND NEWS

Indian Delegation to World Health Assembly

The Government of India's delegation to the Ninth World Health Assembly which opened in Geneva from May 8, 1956, consisted of Dr. A. Lakshmanaswami Mudaliar (Leader), Vice-Chancellor, Madras University, Lt.-Col. C. K. Lakshmanan, Director General of Health Services, Lt.-Col. Jaswant Singh, Director, Malaria Institute of India, Delhi, and Kumari T. K. Adranvala, Chief Nursing Superintendent, Directorate General of Health Services, New Delhi, and Shri K. V. Padmanabhan, Consul General, Geneva, as alternate delegates.

The World Health Assembly is one of the organs of the World Health Organisation. It is composed of delegates representing member-States who meet in regular annual session to determine the broad policies of the organisation, to decide on its programme and its budget and to adopt such international health regulations as may be necessary.

The objective of the Ninth World Health Assembly, *inter alia*, is to study and make recommendations on the general programme of work and determine the scale of assessment of contributions of member-States.

India's Proposals before World Health Assembly

The Government of India has proposed to the World Health Assembly at its 9th session that target dates be fixed for starting wide-based co-ordinated programmes of malaria control leading to world-wide eradication of the disease.

India's proposal points out that this country faces "a colossal problem" of malaria and will arrive only by stages at total eradication. While some areas have been freed from the infection, others remain highly malarious. The danger of re-infection from within the country or from abroad is very great in areas that have been cleared. For this reason India asks WHO for a "critical appraisal of the position of all countries" in South East Asia and other regions similarly situated, and for "fixing target dates for the start and completion of co-ordinated ultra-regional and inter-regional eradication programmes". This, in India's view, is "vitally important, as any tendency to hasty action may lead to irreparably disastrous results."

Another programme item proposed by India requests WHO to call together an Expert Committee on Heart Diseases, and "to initiate necessary action to push forward research and control measures on a world-wide basis."

India has also proposed that minimum uniform qualifications be laid down for doctors on an international basis. This will involve such issues as minimum requirements for accommodation and equipment in medical colleges in relation to the number of admissions, teacher-student ratio and bed strength per student for clinical teaching.

Discussions on the control of leprosy will be based on the recommendation of the WHO Executive Board for more action in this field and a proposal of the Government of Burma calling for a Regional Conference in South East Asia on the subject.

Another mass infection, tuberculosis, will be discussed in a new light following the success of drugs like isoniazid which have made home treatment of tuberculosis patients possible and effective, and brought programmes of TB control within the reach of almost any health administration.

Other mass campaigns to be discussed include those against trachoma and yaws.

The subject for this year's Technical Discussion—which is of an informal character—will be "Nurses: their education and their role in health programmes."

Constitutional matters before the Assembly include some new applications for membership and the question of payment of arrears by the USSR which is resuming active participation in the Organization after several years of inactivity.

International Congress of Otolaryngology

The Sixth International Congress of Otolaryngology will be held at Washington, D.C., U.S.A., on May 5-10, 1957.

Any qualified physician may become a member of the Congress by paying a registration fee of $25.00 U.S.A. Non-medical personnel may register as Associate Members for a fee of $10.00 U.S.A. The registration fee includes the privilege for attending all official meetings of the Congress except the banquet for which an additional charge will be made.

The scientific programme will consist of three Plenary Sessions on Monday, Wednesday, and Friday mornings, May 6, 8 and 10, 1957, at which the following subjects will be discussed: 1. Chronic Suppuration of the Temporal Bone; 2. Collagen Disorders of the Respiratory Tract; 3. Papilloma of the Larynx. These sessions will be opened by internationally recognised authorities and followed by general discussion. Original papers on other subjects will be presented at General Sessions on Tuesday and Thursday mornings and every afternoon, Monday through Friday. Films will be shown at sessions scheduled simultaneously with the General Sessions. Scientific exhibits will be on display during the entire week.

Co-operating with the Sixth International Congress of Otolaryngology, arrangements have been made for several widely recognised postgraduate courses to be given soon after the close of the Congress so that those from outside the United States who attend the Congress can enroll. In each case the number who may register is limited. Special arrangements have been made to reduce considerably the registration fees for those from abroad. Complete information may be obtained from the General Secretary, Sixth International Congress of Otolaryngology, 700 No. Michigan Ave., Chicago 11, Ill., U.S.A.

The Institute of Child Health

The Institute of Child Health, established in 1956, is an academic body recognised by the University of Calcutta for training postgraduate students for the Diploma of Child Health (D.C.H.) of the University. The following courses are available at the Institute:

I. Diploma of Child Health of the Calcutta University (extending over a period of one academic year) begins in June 1956. Only candidates who have taken the M.B.B.S. degree not less than one year previously may be admitted.

II. Specialist Course in Pediatrics extending over a period of 5 years. This will prepare the graduate for taking over independent charge of pediatric institutions or departments of pediatrics and enable him to carry on consultation practice.

III. A Refresher Course for general practitioners extending over a period of 4-6 weeks on special subjects.

IV. D. Phil (Med.) and M.D. Degrees: Arrangements have been made to fulfil the conditions laid down in the Regulations of the University of Calcutta regarding D.Phil (Med.) and M.D. degrees so that students may prepare their thesis under a recognised teacher and sit for these examinations from the Institute.

College of Medicine for Post-Graduate Studies

Calcutta University is now working on a scheme for establishment of a "College of Medicine" which, it is stated, would enable the different medical colleges in Calcutta to provide for post-graduate studies in medicine. And the University had already for the purpose started a post-graduate medical department.

At present, the students have to depend on their research work for post-graduate degrees of the University, but this new scheme would provide them with an opportunity to undertake regular post-graduate studies in medicine.

Medical College in Kanpur

The President, Dr. Rajendra Prasad, laid on April 24, 1956, foundation-stones of two buildings designed to house the Government Medical College and the Cancer Research Institute, to be constructed from private charity.

Cancer Research in N. India

The foundation-stone of the first National Cancer Research Centre in North India was laid on 5 May 1956 by Mr. Deshmukh, Finance Minister, at Safdarjung Hospital, New Delhi. The centre will be attached to the All-India Medical Institute.

The building is expected to cost Rs. 2½ lakhs and the entire amount has been donated by the public. The equipment for the centre will be supplied by the Government which will also maintain the centre.

Speaking on the occasion, Rajkumari Amrit Kaur, Health Minister, said cancer was one of the major problems of the world. The centre would be a nucleus for

research as well as curative work. It would have 50 beds.

Emphasis on Industrial Health

Dr. Harekrishna Mahtab, Governor of Bombay, inaugurating the fourth annual convention of the Society for the Study of Industrial Medicine at the Taj Mahal Hotel, Bombay, on 29-4-56 observed that industrialists should consider industrial health as part and parcel of the industry itself.

He stated that this was very essential as India was on the threshold of an industrial revolution. The Governor expressed the view that the Government, which was the largest employer, should interest itself in industrial health. The time would no doubt come when the State would have to formulate measures for the total health of workers, he asserted.

Dr. M. D. Engineer, in his presidential speech, stated industrial medicine was a social science and it had to line up with other sciences in the interest of the total health of the worker.

He said : "As more and more doctors get trained, we should also have the means to assimilate them in industries. Today this process is pitifully slow. It is heartening to note that the Union Ministry for Labour has urged employers to encourage the development of industrial medical services on sound lines. The Government being the largest employer of labour, it should set an example in this field."

T. B. Hospital near Ranaghat (West Bengal)

The Governor of West Bengal, Dr. H. C. Mookerjee performed the opening ceremony of a T. B. Hospital at Srinathpur village, situated on the outskirts of Ranaghat town in West Bengal by the side of river Churni on April 22 last.

In declaring the hospital open Dr. Mookerjee appealed to people to contribute their mite to the funds for fighting T.B. and to purchase T.B. Sweep tickets. He praised the efforts of the organisers of T.B. Clinic who have courageously started the hospital with 8 beds at present with private help.

Research in Tropical Medicine

Tropical medicine had made it possible to live a much healthier life in hot countries and had a great deal to do in raising the average expectation of life there, said Professor B. G. Maegraith, Dean of the Liverpool School of Tropical Medicine, in an interview in Calcutta on 18-4-56.

Research in tropical medicine, the curative side and the public health side, he said, had made tremendous progress. Effective drugs for the treatment of malaria had been found. He referred to the valuable work done in the Calcutta School of Tropical Medicine on malaria. With the success of the all-India anti-malaria campaign, the incidence of malaria in India had decreased.

Professor Maegraith said there were two types of diseases—metropolitan diseases such as typhoid, which were mostly found in individual cases and diseases such as malaria which were community-wide.

It was possible to fight some diseases such as malaria more or less independently of the population by spreading D.D.T. and taking other measures. But in fighting other diseases such as schistosomiasis (not prevalent in India) co-operation of the people was essential.

Prof. Maegraith is on a five months' tour of Burma, Thailand, Egypt and other countries. While in Thailand he will give demonstrations of the new methods he has developed for resuscitation from the late stage of malaria. A film on the experiment on the basis of which the methods were evolved was shown at the Calcutta School of Tropical Medicine. The methods, in a modified form are also applicable to cholera.

Tuberculosis Conference in Delhi

Speaking at the 17th annual general meeting of the Tuberculosis Association of India on 21-4-56, the Union Health Minister, Rajkumari Amrit Kaur, pleaded that in planning the anti-tuberculosis campaign in the country there should be a harmonious blending of both preventive and curative measures. For this purpose both the administrators and the public had to be educated.

A question that had often been posed in recent times was whether in dealing with T.B. greater emphasis should be laid on preventive measures or on providing facilities for institutional care for tuberculous patients. The authorities were convinced, she said, that in the existing circumstances in India more attention should be paid to preventive work, but simultaneously the other aspect of the T.B. control measures could not be neglected.

She also referred to a resolution recently passed by the Central Council of Health that the control of tuberculosis should be taken up on an all-India basis as a matter of national emergency and in this connexion she urged the T.B. associations in the country to assist the various State Governments in implementing the recommendations of the Council.

Stating that the lack of funds should not stand in the way of carrying out the anti-T.B. campaign, she urged the State Governments to subsidize their T.B. associations in the same manner as the Central Government assisted the Central Association. To find more funds she urged for intensifying the T.B. Seal Sale campaign all over India.

She also announced that an international tuberculosis conference would be held in Delhi early in June 1957. This was the first time that such a conference was to be held in the East.

Rajkumari Amrit Kaur expressed satisfaction at India's achievements in fighting tuberculosis during the last eight years and said that the B.C.G. campaign had come to stay in the country and was progressing satisfactorily in spite of certain difficulties.

Stressing the importance of the voluntary efforts, the Health Minister urged the philanthropists to contribute liberally to carry on the war against T.B. and enable building up of more and more clinics even in the smaller towns.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents

Human Schistosomiasis in India

SIR,—Under the above caption, a paper by Dr. A. K. Varma appeared in your August, 1955 issue on pages 173-175.

The author has quoted certain results of the Indian Council of Medical Research enquiry which require correction. The snail involved in transmission cycle of human schistosomiasis in Gimvi in Ratnagiri district is *Ferrissia tenuis* and this is not an incrimination but has been experimentally proved. Those interested will do well to refer to the following bibliography :—

1. Human Schistosomiasis in India : Discovery of an Endemic Focus in the Bombay State—*Indian J. M. Sc.*, **6** : 760, 1952.
2. Human Schistosomiasis in India : The Study of Snails—*Indian J. M. Sc.*, November, 1955.
3. Human Schistosomiasis in India : Infection of Snails with *Schistosoma hematobium*—*Indian J. M. Sc.*, November, 1955.
4. Human Schistosomiasis in India : Note on Clinical Survey of the Endemic Focus—*Indian J. M. Research* (in the press).
5. Human Schistosomiasis in India : Establishing the life-cycle of *Schistosoma hematobium* in the Laboratory : to be sent for publication—*Indian J. M. Research.*

As the paper appearing in your journal may be quoted here and abroad, I request you to publish this letter soon. I am etc.,

H. I. JHALA,
Officer-in-charge,
Bombay. Schistosomiasis Enquiry, I.C.M.R.
Pathology School, Grant Medical College.

Articles 2 and 3 referred to in the Bibliography were published in October, 1955 issue of *Indian J. M. Sc.*, and not in November, 1955.—ED., *J. Indian M. A.*

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SIR,—I am thankful to the correspondent for the favour of furnishing some references on the subject, some of which are still in the making. I also note with much interest that the snail-intermediary of human schistosomiasis in Gimvi (Ratnagiri district) has now been experimentally proved and that the paper is under construction for publication. In support of his criticism the correspondent cites a reference which appeared in the press much later than my paper. In all fitness of things, I hope you will agree that the authors of the subsequent publications should refer to previous works in their papers themselves and it is hardly necessary or desirable to come out with any statement like "and this is not an incrimination but has been experimentally proved" in a letter form as suggested by the correspondent. I am etc.,

A. K. VARMA, PH.D. (LOND.),
Patna. Livestock Research Station, Bihar.

Retrospective Recognition of Medical Qualifications and Maintenance of Standards

SIR,—It is with interest one reads Da. C. S. Patel's objection to the decision of the Government of India in recognising the State Medical Faculty of West Bengal (M.M.F.) and the M.B.B.S. of the Universities of Bihar, Poona, Utkal, Gujarat, Nagpur and Rajputana, retrospectively from the date of their institution (*J. Indian M. A.*, **26** : 332, 1956). His anxiety in maintaining high standards in medical colleges is laudable. But efforts of the Medical Council will be ineffectual, if they think of preservation of standards by only giving recognition or withholding it. The institution of post-graduate studies and intensive research work in the colleges will create a better order of things. Such possibilities are lacking in some institutions which have been recognised by the Medical Council of India—for example, the Mysore Medical College which came into existence thirty-two years ago has not been able to grow into adulthood, as yet. So far as the M.B.B.S. of Mysore University is concerned, the Medical Council is at fault on another score. The reciprocal arrangements for recognition of medical qualifications, to which Dr. Patel refers, seem to be null and void in its case. A medical graduate of the University of Mysore cannot seek election to membership of the British Medical Association because that qualification is not registrable on the permanent register in that country, even though there are reciprocal arrangements for recognition of qualifications granted in some other Universities in India and U.K.

In the context of such a state of affairs, it is advisable that the Medical Council will put its house in order and speak from a "position of strength". I am etc.,

Kasturba Medical College, (DR.) M. S. NILAKANTA RAO,
Mangalore. B.SC., M.B.B.S.

REVIEWS

Vital Statistics Special Report on Health and Health Services in Rural and Urban Areas of West Bengal, State Volume, 1954. Edited by Dr. P. G. Chowdhury, Asst. Director of Health Services (Vital Statistics), West Bengal; Government of West Bengal, Directorate of Health Services, Office of Vital Statistics; 44 × 28 cm., 7+80 folios; Price not mentioned.

In spite of their being much maligned, statistics undoubtedly have their value, especially in planning for the future. This aspect of statistics has been emphasised in the Foreword contributed by Dr. A. D. Mukharji, Minister in charge of the Medical and Public Health Department of the West Bengal Government, in the Introduction by the then Director of Health Services, and in the Preface by Dr. Chowdhury. Consisting of many tables, charts and diagrams in colour the material covers a wide field. The first three sections are devoted to general statistics of West Bengal, the world and India. The next three sections deal with demographic changes, agriculture and education. The next subject dealt with

is finance with special reference to health. The largest section, that on medical institutions follows. Health personnel, hospital services, vital statistics, water supply, roads, community projects, fairs and festivals and progress of medical institutions complete the volume.

Altogether the volume under review is a valuable addition to our statistical literature. One thing that emerges clearly from the volume is the great progress West Bengal has made in the field of medical and public health. It is clearly ahead of other States in India.

We wish we had more recent statistics to deal with, for statistics lose much of their value when not up-to-date.

The printing is quite good though the choice of colours has not always been happy. We have found only one misprint and a discrepancy. The name of the editor appears as "Chowdhury" on the cover and as "Choudhuri" at the end of the preface.

Journal of the Anatomical Society of India (Vol. IV, No. 2, December 1955)—Edited by L. W. Chacko, M.B., B.S., M.SC., D.PHIL. (OXON.), from the Department of Anatomy, Christian Medical College, Vellore (N.A.), South India.

This journal, with 48 pages of reading matter, contains quite a few interesting articles, out of which special mention may perhaps be made about the following two—"The development of the nerve supply of the human oesophagus and stomach" and "The lateral geniculate body in the gibbon". Several articles in this number show a reasonable amount of experimental work being carried on, in different aspects of anatomy in various centres of learning in India. This augers well and we hope to see records of further progress in the subject.

Atlas of General Surgery—By Dr. Joseph R. Wilder, M.D. Published by C. V. Mosby Company, St. Louis, Missouri, U.S.A., 1955. Board bound, 14″ x 10¼″, 221 pages with 101 plates. Price $13.50.

Extensive and rapid advances in the medical sciences, specially in the art and science of surgery, are now-a-days generally reflected in the continuous attempts, all the world over, to enlarge and broaden the scope of general surgery. The steadily increasing knowledge again is in itself a constant challenge to the surgeon who has to be thorough and meticulous about the care of the patients during the pre-operative and postoperative periods, besides carrying out a skilful surgical procedure. This book is intended to emphasise selected techniques for practical work in general surgery—found most satisfactory by the author. These techniques have been explained briefly, along with a mention of the difficulties and probable complications. Besides there are many elucidating illustrations. Great care seems to have been taken to make the illustrations practical, to the point and of easy appreciation of the exposures and anatomy as evidenced in the operating room. This book will serve as a practical and useful guide for reference by residents, house-officers, students and also by young surgeons undertaking independent operative procedures.

OBITUARY

Dr. P. N. Ghose

Dr. P. N. Ghose, the President of the Dibrugarh branch of the Indian Medical Association since its inception, breathed his last on 12 April 1956.

Born on 20th June 1895, Dr. Ghose passed his early student life at Shillong. He passed the M.B. examination from Calcutta Medical College in 1919. He set up practice in Dibrugarh and soon became a very successful physician.

Dr. Ghose did not keep himself confined to his profession only. He was deeply interested in various social and cultural activities. The Government conferred on him 'Rai Bahadur' for his civic work.

DR. P. N. GHOSE

He was President of Assam State Branch of Indian Medical Association for 1953; member, Indian Medical Council from 1954; Chairman of the Managing Body of Indian Red Cross Society, Dibrugarh District; member, governing body, Assam Medical College and Member of Faculty of Medicine, University of Gauhati.

Dr. Ghose earned a good fortune, but money was of secondary importance to him, the primary being service to the ailing and suffering humanity.

Dr. Ghose was a native of Naihati (Hoogli), West Bengal, but he adopted Dibrugarh as his own town and Assam and its people as his very own. He donated his palatial house to the Government of Assam for establishing a public welfare institution

Dr. Ghose left Dibrugarh *en route* to New Delhi to attend a meeting of I. M. Council on 4-3-56, but fell seriously ill at Calcutta. Following an abdominal operation on 8-4-56 he passed away on 12-4-56.

May his soul rest in eternal peace!

SUPPLEMENT
Journal of the Indian Medical Association

VOL. 26, No. 11 CALCUTTA JUNE 1, 1956

XX U.P. STATE MEDICAL CONFERENCE, BAREILLY

The twentieth U. P. State Medical Conference was held at Bareilly on 27-28 November 1955. Dr. B. K. Saxena, Chairman, Reception Committee, accorded a hearty welcome to the delegates and guests. In his address, he narrated the history of Bareilly, its political and cultural importance. He also discussed in short the available medical facilities and referred to the shortcomings regarding health conditions of the people.

Dr. Durga Prasad who presided over the conference, in course of his address said :

"The history of Indian Medical Association and its U.P. State Branch is not very remote. The Association may be considered still in a formative stage when we bear in mind that this is only the 20th State Conference. The development of an organisation depends upon its numerical strength as well as upon the efforts made by its members for the promotion of its prosperity and well-being. We find that even today there are far more members of our profession outside the Association than inside it. It is the duty of every one of us to find out causes as well as remedies for this state of affairs. Advantages and privileges of being members of Indian Medical Association must be brought home to non-members. It will be my earnest endeavour during my year of office to form many more new branches and to bring several more non-members of our profession to join the Association. In U.S.A. and England we hardly come across a qualified doctor who is not a member of the Associations of these countries.

QUACKERY

My predecessors in office, year after year have been bringing to the notice of authorities the existence of quackery on a large scale. Many methods have been suggested to eradicate it, but quackery goes on. Every town is full of quacks. Unqualified persons have been using antibiotics, sulpha and other drugs unhampered, leading to loss of lives of hundreds of patients. Use of these drugs by unqualified persons should be made a criminal offence and a Law should be passed to this effect. Compulsory birth and death certificates by registered medical practitioners would to a large extent check quackery. I hope the State Government will come forward with these Laws.

OTHER SYSTEMS OF MEDICINE

Much has been said in the past regarding other systems of medicine. I have my own views regarding them. I can only say on this occasion that Science is progressive. Every branch of Science has constantly been developing. During the last quarter-century Medical Science has made a remarkable progress and has completely revolutionised the treatment of diseases. To call the modern scientific system of medicine as western would be a complete misnomer. Almost all the nations of the world, including India, have contributed to make it develop to its present stage. If Astrology, Mathematics, Physics, Chemistry and other liberal Arts and Sciences cannot be called western, why should modern scientific system of medicine be singled out for this purpose? Science is not the property of a single nation. It is universal. Our aim is to alleviate the human sufferings and to save human lives. We must do so by the best means at our disposal. I do not find fault with the Ayurvedic and Unani Systems of medicines, except for the fact that they have remained more or less static for centuries.

GOVERNMENT HOSPITALS AND DOCTORS

It is a matter of great regret that our countrymen have not yet realised the value of service they can render to nation by founding well-equipped and modern hospitals. Well-to-do and philanthropic people should give this problem their serious thought. We raise memorials

DR. DURGA PRASAD, THE PRESIDENT OF THE CONFERENCE.

to perpetuate the names of our nation-builders and patriots, our relatives and friends. What better memorial can we have than to build a modern hospital well-equipped with modern scientific appliance where the poor could be treated free and those who can afford to pay, on a reasonable payment? Kamala Nehru Hospital in Allahabad is a living example of such a memorial. For the present we have to depend almost entirely on Government hospitals.

In rural areas, hospitals are few and far between. Desirability of medical relief to rural population cannot be overemphasised. Government plan of compulsory

service by a medical graduate for a certain period in rural areas has been discussed. It has been suggested that stipends and scholarships be given to medical students on an undertaking that they would serve in villages for a certain period. Better housing and other facilities and more liberal grants to medical practitioners desirous of settling in rural areas have been suggested. All have their relative values and the Government is competent to decide a particular method suitable to a particular locality. But till such time that the whole of rural population can get medical service by qualified practitioners a number of mobile units must be set up. National Health Service should be established in rural areas with Health Assistants in charge of groups of villages who should work in contact with fully trained physicians. This is also envisaged in second five year plan.

Certificates

It has been a grievance of private medical practitioners that certificates given by them to Government servants are often required to be countersigned by the Civil Surgeon. Every medical practitioner is not dishonest and all the Civil Surgeons are not honest. A doctor who is allowed to treat a patient must also have the privilege of issuing certificates to his patients without the necessity of counter-signature. This restriction must be removed.

Second Five Year Plan

In preparing the first Five Year Plan the Government had not found it worthwhile to consult the Indian Medical Association and eminent physicians and surgeons of the country regarding medical and public health problems. There was no definite outline and no definite aim as far as 'health' was concerned. As a result Medical and Public Health Services did not benefit much from the first Five Year Plan. There has certainly been improvement in the prevention of Malaria by the adoption of vigorous antimalarial measures, but no encouraging results have been obtained in other spheres of public health. In second Five Year Plan more Health Services are to be created. The number of dispensaries and hospitals and facilities for medical training is to be increased in rural areas. The number of hospital beds is to be increased by 100 per cent and that of registered doctors to be employed by 29 per cent. Indian Medical Association has prepared a comprehensive plan covering all Medical and Public Health problems. I hope the Planning Department of the State Government will make a careful study of this plan and will implement the recommendations within the frame-work of Second Five Year Plan.

It would not be out of place for me if at this stage I brought a few things to the notice of the Government. One of them is the imposition of Sales Tax on medicines and other accessories like X-Ray, Ultra-violet Rays, etc. This Sales Tax should be abolished as ultimately it affects poor suffering humanity. There is also a great demand for properly qualified nurses and compounders. The Government should take up training of these in Medical Colleges and District Hospitals more vigorously.

Family Planning

Government efforts have touched only a fringe of problem of wide-spread ill-health in the community. This is accompanied by a low standard of living, and even then the growth of population has been striking. Within the three decades 1891-1921 the inhabitants of the parts now included in Republic of India increased by about 16 millions, while during the next thirty years 1921-1951, the corresponding increase was about 105 millions. With the use of antibiotics and other modern drugs on the one hand and higher standard of living on the other and with the policy undertaken by the State to reduce sickness and mortality and to prolong life, a simultaneous effort to bring down the birth rate becomes a matter of fundamental importance. The Public Health Department can to a large extent help bring about contact with homes of the people through doctors, health officers and maternity and child-welfare centres and their health-visitors in order to educate them in methods of family planning. Branches of Indian Medical Association throughout the State should be entrusted with the task of opening family planning clinics and educate the community by carrying on an extensive propaganda through various means such as public lectures, cinema-slides, newspaper articles, etc.

Food and Drug Adulteration

Adulterated food is the greatest problem of the present time. This is an acknowledged fact that diseases find their way when proper food is not available. Offenders in food and drug adulteration must be given an exemplary punishment. Expectant mothers belonging to the poor class should be supplied with pure milk by the State. School-children should be given free tiffin and there should be a periodical check up of their health.

Medical Education

Very recently a Medical Education Conference was held in New Delhi which was presided over by Rajkumari Amrit Kaur, the Union Health Minister and inaugurated by Dr. B. C. Roy. Prime Minister Nehru also spoke at the Conference which was attended by Deans of all Medical Colleges in the country, Members of the Executive Committee of the Indian Medical Council and observers from the World Health Organisation, Rockfeller Foundation and Technical Co-operation Mission and representatives from several other countries. The conference of such a distinguished type must have discussed Medical Education in all its bearings and they must have evolved a system of Medical Education suitable to the present needs of the country. I want to bring to the notice of Government and Universities want of facilities for Post-graduate training and Research. We have enough brain in the country but opportunity is denied to our young graduates. Fellowships must be awarded to the deserving graduates and I hope facilities for Post-graduate training and research will be provided far more liberally at our colleges where specialised training in ali branches of Medicine should be given. The Government and other bodies should rank the Post-graduate Degree and Diplomas granted by our Universities and Colleges on the same level as the equivalent Diplomas of foreign countries. This difference should specially be abolished in the personnel of government services. There should also be provision for a short Refresher Course at the Medical Colleges both for private medical practitioners and government servants where doctors could go for a short period and brush up their medical knowledge.

B. C. G. Vaccination

Recently there has been a big controversy in the Press regarding the efficacy and desirability of B. C. G. vaccination. Intensive experiments on this have been carried out in Scandinavian countries and Tuberculosis in these countries has practically disappeared. Eminent doctors in our country have from time to time demonstrated harmlessness and benefits of these vaccinations. Hundreds of thousands of children as well as adults have been tested and lacs have been inoculated. A few deaths here and there are the results of some extraneous factors. Indian Medical Association should carry on intensive propaganda in favour of the B. C. G. vaccination so that when vaccination team next visits a particular locality people may be ready to avail themselves of the opportunity.

Ap Ka Swasthya

A beautiful Hindi Health Magazine "Ap Ka Swasthya" is being published from Banaras every month under the auspices of U.P. State Branch of Indian Medical Association. It is our own magazine and I strongly recommend it not only to the general public but also to doctors. Our members should not only subscribe it but should contribute articles of health and public interest in order to make it more useful.

Medical Ethics

May I now be permitted to speak a few words to my professional brothers and sisters. The practice of medicine brings us in contact with patients, with our colleagues and on several occasions with public opinion and public interests. In each of these relations we have often to face situations not always free from difficulties. These relations have evolved certain practices which we call Medical Ethics and etiquette. Our first consideration in professional code should be recognition of our responsibilities to the patient and no demand or consideration should take precedence of this duty. It is often suggested that medical practitioners are guided in this regard by selfish motives touching their own pockets. But we have to live, and as long as we have the welfare of the patient as our concern consistent with the remuneration we get for our services we are doing our duty and not violating our professional code of ethics.

I make a special appeal to senior members of our profession to safeguard the interests of junior members. They should set an example before them of correct professional conduct. Intra-professional relations at times create problems which may easily be the cause of strained relations between colleagues. With a sense of good fellowship, a recognition of common difficulties and practice of frankness most disputes can be resolved. May I suggest that one meeting of every branch of Indian Medical Association each year be devoted entirely to the discussion of Medical Ethics. It will create a sense of duty among members towards their patients, their colleagues and the community.

In this connection I also suggest one more thing which has been uppermost in my mind and this is with regard to the treatment of our colleagues and their dependent relations when they are ill. It is the experience of most of us that they are usually neglected and do not get the attention they deserve. They should in fact be given our best attention when we are called upon to treat them. It should be considered as our professional duty to the same extent as if we are treating a paying patient. On the other hand it has also been found that doctors when they fall ill do not conduct themselves as patients and make a muddle of their treatment by entertaining usually available advice of their numerous medical friends who come to visit them. They should select their doctors for treatment and stick to him just as they expect their patients to do. If a second opinion is wanted, the doctors incharge will obtain it as he does in case of any other patient."

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The following resolutions were passed at the Conference :

1. This Conference places on record its deep sense of sorrow at the sad demise of the following members of the profession and conveys its heartfelt sympathy with the members of the bereaved families :—

1. Dr. M. S. Varma, Allahabad; 2. Dr. B. P. Mital, Allahabad; 3. Dr. Shyam Swaroop, Bareilly; 4. Dr. R. N. Lal, Mussorie; 5. Dr. N. Man Singh, Kanpur; 6. Dr. B. N. Saigal, Lucknow; 7. Dr. Ved Prakash, Bijnor; 8. Dr. H. L. Dubey, Meerut; 9. Dr. Dev Rishi Sharma, Muzaffarnagar; 10. Major Hans Raj Kapur, Agra.

2. This Conference welcomes the suggestion as contained in the Municipal Corporation Draft Bill to give special representation for Medical and Public Health workers as aldermen but suggests that they should be nominated on the recommendation of the Local Branches of the Indian Medical Association.

3. Resolved that the State Government should be requested to make arrangements of providing a well equipped pathological laboratory with qualified whole time staff in every district. This should be free for the poor on the advice of the attending registered medical practitioner and on nominal charges for others. Further resolved that where facilities exist at present, it should be extended as soon as possible.

4. This Conference suggests to the U.P. State Government that the plan as submitted by the U.P. State Branch of the Indian Medical Association may be taken into consideration while finalising the 2nd Five Year Plan on Medical and Public Health development of the State.

5. This Conference resolves that in the interest of the National health, it is necessary that the members of the Indian Medical Association should take an active interest in giving prophylactic inoculations for those infectious diseases for which efficient prophylactics are available and, therefore, the Conference recommends to all its Branches to organise prophylactic measures on their own resources and try to educate the public so that they may appreciate the value of such measures.

Further resolved that the Government should organise prophylactic measures in collaboration with the local Branches of the Indian Medical Association.

6. Resolved that the Universities in U. P. be requested to start courses in Anatomy, Physiology and Biochemistry for B.Sc. and post-graduate courses.

7. This Conference recommends to the U. P. Government that for the purposes of uniformity and equity, the conditions of service, pay and emoluments should be the same in all Medical Colleges in U.P. in similar departments.

8. This Conference resolves that there should be one virus research Centre at some suitable place in the State.

BRANCH NOTES

AJMER BRANCH—A meeting was held on 31-3-56. Forty-six members were present. Office bearers for 1956-57 were elected with Dr. S. P. Wanchoo as president, Dr. B. M. Sharma as hony. secretary and Dr. A. Sahai as hony. joint secretary. The annual report for 1955-56 and the audited accounts were adopted.

ALIGARH BRANCH—A meeting of the branch was held on 4-2-56 with Dr. D. P. Sharma in the chair. Thirty members and forty guests were present. Dr. Mohan Lal, Chief Medical Officer, Gandhi Eye Hospital, Aligarh was congratulated on his obtaining the title of Padam Shri in recognition of his meritorious services.

ALLAHABAD BRANCH—A scientific meeting of the branch was held on 21-1-56. Dr. J. C. Mehta of Northern Railway, spoke on "Jaundice". Dr. S. N. Chatterjee, Civil Surgeon, Allahabad demonstrated 2 interesting cases of foreign body in stomach.

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A clinical meeting was held on 15-2-56. Dr. J. C. Mehta demonstrated 4 interesting cases. One case of gun shot wound in the abdomen was discussed by Dr. S. N. Chatterjee.

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A meeting of the branch was held on 3-3-56. Dr. A. K. Ghosh read a paper on Common Laboratory Test.

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A social gathering was arranged on 24-3-56. Sixty-four members were present. Members enjoyed the function thoroughly.

BANARAS BRANCH—At a meeting of the branch held on 25-2-56 the members condoled the death of Dr. C. M. Bose on 19-2-56, an old member of the association.

BERHAMPORE BRANCH —The annual meeting of the branch was held on 8-4-56. Office bearers were elected for the current year with Dr. G. N. Sahu as president, Dr. M. V. Ramanayya as secretary and Dr. R. Krishna Rao as joint secretary.

CHITALDROOG BRANCH—A clinical meeting of the branch was held on 18-2-56 with Dr. N. V. Puttabhatta in the chair. Dr. Jayaseela Reddy read a paper on the treatment of "Unalgin in painful musculo skeletal condition". Dr. Rajeswari read a paper on "Tumour Abdomen"; Dr. K. S. Devendra read a case report on acute "Diarrhoea"; Dr. D. Shamanna spoke on "Tuberculosis"; Dr. Jyothi read a case report on "Cancer Liver". Health film was shown to the students and the public. The president emphasised the importance on the prophylactic aspect than the curative.

COIMBATORE BRANCH—The annual general body meeting of the branch was held on 11-3-56 with Major R. S. Rao in the chair. One hundred and thirty doctors were present. Dr. Rao inaugurating the annual day celebrations pleaded for protected water supply for the city of Coimbatore. Dr. A. Venugopal of General Hospital, Madras gave a talk on "Calculi in the Genito Urinary Tract". Office bearers for 1956 were elected with Dr. T. V. Sivanandam as president, Dr. S. C. Rajarathnan as secretary and Dr. C. N. Santhanam as associate secretary. Dr. Jacob Chandy of Christian Medical College, Vellore gave a talk on "Head Injuries". Dr. C. Satyanarayana of Stanley Hospital spoke on "Cancer of the Larynx".

DEORIA BRANCH—The general meeting of the branch was held on 8-4-56. Dr. S. N. Mukherji presided. Six members were present. The proceedings of the last meeting were confirmed after slight corrections. Letters from the State branch were considered. Dr. R. M. Singh showed a case of "Dermal Leishmania" which was wrongly diagnosed as leprosy. Dr. S. C. Acharya read a paper on "Hodgkin's Disease" citing 2 cases.

DHANBAD BRANCH—The branch observed Health Week from 12-18 March 1956. General sanitation, disinfection of water sources, mass inoculation against cholera and vaccination were carried out. Public meetings were held at different places and lectures were given on clean habits, general sanitation and prevention of diseases. A baby show was organised at Poddardih and it was very successful.

DUM DUM BRANCH—A meeting of the branch was held on 25-3-56 with Dr. P. C. Ghosh in the chair. Fourteen members were present. Office bearers were elected with Capt. P. C. Ghosh as president and Dr. P. M. Roy and Dr. N. L. Sen Gupta as hony. secretaries. Congratulations were accorded to the 5 members of the medical profession who have been elected commissioners of the Dum Dum and South Dum Dum Municipalities.

FEROZEPORE BRANCH—A meeting of the branch was held on 2-4-56. Dr. Raja Ram Bhola presided. Twenty-five doctors were present. Dr. K. M. Singh, Civil Surgeon, spoke of "Heart Failure and its Management". Dr. M. E. Tucker of Ludhiana Medical Collegt spoke on "X-ray Findings in diseases of Heart."

GULBARGA BRANCH—A meeting of the branch was held on 19-2-56. Dr. L. R. Despande presided. Dr. B. V. Mulay, ex-president, I.M.A.' spoke on "Changing Trends in Therapeutic Principles". He stressed on abuse of Antibiotics and requested the medical men to be more rational.

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A meeting of the branch was held on 18-3-56 with Dr. L. R. Despande in the chair. Dr. Despande spoke on "Diabetes and its treatment".

GORAKHPUR BRANCH—A meeting of the branch was held on 21-1-56 with Dr. K. N. Srivastava in the chair. Thirty-four members were present. Dr. S. S. Varma cited a case of "Hyperthyroidism" in a lady where Thyroidectomy was done to give relief to the patient. Dr. N. M. Seth of Sitapore Eye Hospital cited a case of "Laurence Moon Beedle's Syndrome" in a boy of 8 years.

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A meeting of the branch was held on 24-3-56. Dr. C. M. Aga of Railway Hospital presided. Thirty-five members were present. The meeting condoled the death of Dr. R. S. Lal, Superintendent, Mental Hospital, Agra. Rs. 250/- was contributed to the building fund, Central Office, I.M.A. The following cases were cited, (1) Primary Concomitant Alternate Divergent Strabismus and (2) Herpes Zoster by Dr. G. K. Sharaf, (3) Tubal Tumour in a lady of 30 by Dr. R. Pd. Misra, (4) Umbilical Hernia in a lady of 20 by Dr. R. Pd. Mathur and Dr. Misra, (5) Twisted Ovarian cyst by Dr. R. Pd. Mathur. A paper on Medical aspects in Russia was read by Dr. S. P. Agarwal.

GURDASPUR BRANCH—A meeting of the branch was held on 12th November 1955 under the chairmanship of Dr. S. Singh, Civil Surgeon. Nineteen doctors attended. Dr. W. T. B. McAlister showed and discussed some interesting cases. He also read a paper on "Peptic Ulceration : a ubiquitous problem".

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A combined meeting of the Gurdaspur and Pathankot branches was held on 17th December 1955 under the chairmanship of Dr. S. Singh, Civil Surgeon, Gurdaspur. Dr. B. Sachdeva, Medical Officer I/C Tuberculosis Clinic, Gurdaspur read a paper on "An Insight into BCG Vaccination" and Dr. H. R. Khera of Pathankot on "Puerperium and its management".

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A meeting of the branch was held on 14th January 1956. Dr. S. Singh, presided. Nineteen doctors attended. Dr. P. N. Chhabra, Assistant Surgeon, Gurdaspur read a paper on "Jaundice".

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A meeting of the branch was held on 19th February 1956 under the chairmanship of Dr. S. Singh. Twenty-one doctors attended. Dr. (Miss) Shanti Sachdeva read a paper on "Role of Pitutrin injection in Maternity cases". Dr. M. Singh discussed some latest advances in diseases of children. Dr. B. S. Sachdeva exhibited some X-ray skiagrams.

JAGATDAL BRANCH—At a meeting held on 1-5-56 the members condoled the unnatural and tragic death of Dr. S. C. Bhattacharya. Dr. Bhattacharya was murdered in his banglow by unknown persons.

JAYASINGPUR BRANCH—A meeting was held with Dr. B. T. Burse in the chair. Office bearers were re-elected with Dr. B. T. Burse as president and Dr. F. A. Deane as hony. secretary. During the last year, the branch held eleven general meetings and one executive committee meeting.

JEHANABAD BRANCH—"Health Week" was celebrated from 12-3-56 to 18-3-56. Mass vaccination and sanitation drive, public utility lectures and baby shows were arranged. Twenty-six prizes of Rs. 40/- each were awarded to children of good health.

KALYANI BRANCH—A special meeting of the branch was held on 30-1-56 with Dr. B. Mukherjee in the chair. Eighteen members were present. Dr. A. R. Dutt, the secretary of the branch was given farewell on his transfer from Kalyani. A group photograph was taken.

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A meeting of the branch was held on 15-2-56 with Dr. B. Mukerji in the chair. Dr. K. R. Adhikary was elected as secretary of the branch in place of Dr. A. R. Dutt, transferred.

KASHIPUR BRANCH—At a meeting of doctors of Kashipur, a branch was formed with Dr. R. Ahuja as president and Dr. R. B. Singhal as hony. secretary. It was decided that monthly meetings will be held on the first Sunday of every month.

KHANDWA BRANCH—The annual election of the branch took place in November 1955 and office bearers were elected with Dr. R. S. Verma as president and Dr. D. D. Gokhale as secretary.

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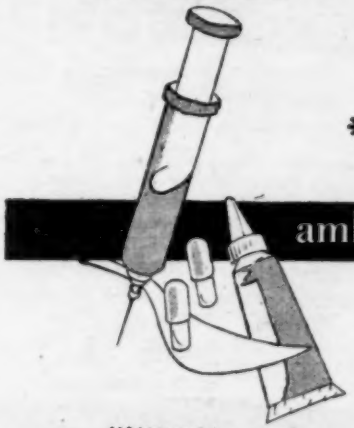

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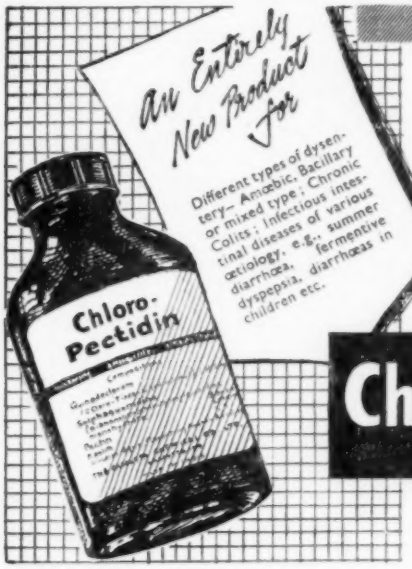
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